

# **STIC Search Report**

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**STIC Database Tracking Number: 130657**

**TO: John Pak**  
**Location: 4a25 / 4c70**  
**Tuesday, August 31, 2004**  
**Art Unit: 1616**  
**Phone: 272-0620**  
**Serial Number: 10 / 070486**

**From: Jan Delaval**  
**Location: Biotech-Chem Library**  
**Rem 1A51**  
**Phone: 272-2504**

**jan.delaval@uspto.gov**

### **Search Notes**

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**Enter your Contact Information below:**Name: Employee Number: Phone: Art Unit or Office: Building & Room Number: Enter the case serial number (Required): 

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Include the country name and patent number.
- Provide examples or give us relevant citations, authors, etc., if known.
- FAX or send the **abstract, pertinent claims** (not all of the claims), **drawings, or chemical structures** to your EIC or branch library.

Mailbox  
REM 4C70

Jan 8/3/04

**Enter your Search Topic Information below:**

I. Method of treating sepsis by administering a composition wherein

1).lipid provides greater than 35% of the total energy of the composition,

2) ratio of n-6 fatty acid to n-3 fatty acid is between 2 and 7 (that is to say, n-6 is present at 2 to 7 times higher amount than n-3 fatty acids).

II. Same method as above, but with an additional feature: 25-70% of the lipids in the composition is from MCT (medium chain triglycerides) and there is less than 15% by weight saturated fatty acids, not counting the MCTs.

n-6 fatty acids (also known as omega-6 fatty acids): linoleic acid, gamma-linolenic acid, dihomo-gamma-linolenic acid, arachidonic acid, docosadienoic acid

n-3 fatty acids (also known as omega-3 fatty acids): EPA (eicosapentaenoic acid), DPA (docosapentaenoic acid), DHA (docosahexaenoic acid), ALA (alpha linolenic acid).

**Special Instructions and Other Comments:**

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Last Modified: 08/20/2004 10:04:50

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(FILE 'HOME' ENTERED AT 09:35:24 ON 31 AUG 2004)  
SET COST OFF

FILE 'HCAPLUS' ENTERED AT 09:35:33 ON 31 AUG 2004

L1 1 S (WO2000-EP8731 OR EP99-118173)/AP,PRN  
E NESTLE/PA,CS  
L2 2617 S NESTLE?/PA,CS  
E TURINI M/AU  
L3 25 S E3-E6  
E ROESSLE C/AU  
L4 8 S E3,E4  
E ROSSLE C/AU  
L5 7 S E3,E4  
E BREUILLE D/AU  
L6 34 S E3,E4  
E CROZIER W/AU  
L7 5 S E3,E9,E10  
E WILLI G/AU  
E CROZIER G/AU  
L8 24 S E3-E8  
E FINOT P/AU  
L9 80 S E4-E7  
E RICHELLE M/AU  
L10 45 S E3,E4,E8  
E DUTOT G/AU  
L11 11 S E3,E5

FILE 'REGISTRY' ENTERED AT 09:45:51 ON 31 AUG 2004

L12 6 S (LINOLEIC ACID OR  $\Gamma$ -LINOLENIC ACID OR DIHOMO- $\Gamma$ -LI  
L13 9 S (EICOSAPENTAENOIC ACID OR DOCOSAPENTAENOIC ACID OR DOCOSAHEXA  
L14 1 S 92661-11-5  
L15 7 S L12,L14  
L16 1 S DIHOMO- $\Gamma$ -LINOLEINIC ACID/CN  
L17 7 S L15,L16

FILE 'HCAPLUS' ENTERED AT 09:52:10 ON 31 AUG 2004

L18 52030 S L17  
L19 1471 S L17(L) FFD/RL  
L20 1280 S L17(L) THU/RL  
L21 20593 S L18 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
L22 63109 S (ARACHIDONIC OR LINOLEIC OR GAMMA LINOLENIC OR DIHOMO GAMMA()  
L23 24880 S L22 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
E FATTY ACIDS/CT  
E FATTY ACIDS (L) N/CT  
E FATTY ACIDS (L) OMEGA/CT  
E FATTY ACIDS (L) POLYUNSAT/CT  
L24 2403 S E13,E16  
L25 2519 S FATTY ACID?/CT (L) (N6 OR (N OR OMEGA) () 6 OR OMEGA6)  
L26 276 S L24,L25 (L) FFD/RL  
L27 168 S L24,L25 (L) THU/RL  
L28 2167 S L24,L25 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?  
L29 30232 S L19,L20,L21,L23,L26,L27,L28  
L30 26049 S L13  
L31 1830 S L13 (L) FFD/RL  
L32 1316 S L13 (L) THU/RL  
L33 14009 S L30 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
L34 12812 S (EICOSAPENTAENOIC OR DOCOSAPENTAENOIC OR DOCOSAHEXAENOIC OR A  
L35 8135 S L34 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
E FATTY ACIDS (L) POLYUNSAT/CT  
L36 5489 S E12,E14,E15  
L37 5703 S FATTY ACIDS?/CT (L) (N3 OR (N OR OMEGA) () 3 OR OMEGA3)

L38 855 S L36,L37 (L) FFD/RL  
 L39 654 S L36,L37 (L) THU/RL  
 L40 5131 S L36,L37 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?  
 L41 17717 S L31,L32,L33,L35,L38,L39,L40  
 L42 11526 S L29 AND L41  
 L43 21471 S L18-L29 AND L30-L41  
 L44 5431 S L43 AND LIPID#/CW  
     E MEDIUM CHAIN TRIGLYCERIDE/CT  
     E E11+ALL  
 L45 1538 S E2  
 L46 2489 S ((MED OR MEDIUM OR M) ()CHAIN) (L) (GLYCERIDE OR TRIGLYCERIDE)  
     E FISH OIL/CT  
     E E5+ALL  
 L47 4257 S E2  
 L48 1570 S L47 (L) (FFD/RL OR THU/RL)  
 L49 3744 S L47 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
 L50 715 S L47-L49 AND LIPID#/CW  
 L51 85 S L47-L49 AND L45,L46  
 L52 5950 S L44,L50,L51  
     E SEPSIS/CT  
 L53 7317 S E3,E5-E10  
     E E8+ALL  
 L54 8136 S E1,E2,E3  
     E E3+ALL  
 L55 3750 S E4+NT  
     E E3+ALL  
 L56 10652 S E3+NT  
     E SHOCK/CT  
 L57 4729 S E4-E12  
     E E4+ALL  
 L58 13891 S E8,E9,E7+NT  
     E E26+ALL  
 L59 6857 S E3,E2+NT  
 L60 8 S L52 AND L53-L59  
 L61 152 S L1-L11 AND L18-L60  
 L62 17 S L61 AND L47-L49  
 L63 55 S L61 AND L42,L43  
 L64 30 S L63 AND LIPID  
 L65 41 S L62,L64  
 L66 23 S L62,L63 NOT L65  
 L67 8 S L52 AND ?SEPSI?  
 L68 13 S L52 AND ?SEPTI?  
 L69 17 S L60,L67,L68  
 L70 1 S L61 AND L69  
 L71 14 S L69 AND (PY<=1999 OR PRY<=1999 OR AY<=1999)  
 L72 3 S L69 NOT L70,L71  
 L73 1 S L72 AND SEPTIC RAT  
     SEL DN AN L71 2 6 12 13  
 L74 10 S L71 NOT E1-E12  
 L75 11 S L73,L74,L70  
 L76 11 S L75 AND (N6 OR N 6 OR OMEGA6 OR OMEGA 6 OR N3 OR N 3 OR OMEGA  
 L77 8 S L76 AND (LINOLEIC OR ?LINOLENIC? OR ARACHINDONIC OR DOCOSADIE  
 L78 11 S L75-L77 AND L1-L11,L18-L77

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FILE COVERS 1907 - 31 Aug 2004 VOL 141 ISS 10  
FILE LAST UPDATED: 30 Aug 2004 (20040830/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all hitstr tot 178

L78 ANSWER 1 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 2001:208096 HCAPLUS  
DN 134:236858  
ED Entered STN: 22 Mar 2001  
TI High lipid diet for prevention or treatment of sepsis  
or inflammatory shock  
IN Turini, Marco; Roessle, Claudia; Breuille,  
Denis; Crozier-Willi, Gayle; Finot, Paul-Andre;  
Richelle, Myriam; Dutot, Guy  
PA Societe des Produits Nestle S.A., Switz.  
SO PCT Int. Appl., 29 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
IC ICM A61K031-00  
CC 18-5 (Animal Nutrition)  
Section cross-reference(s): 1, 17, 63

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001019356	A2	20010322	WO 2000-EP8731	20000907 <--
WO 2001019356	A3	20010517		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1090636	A1	20010411	EP 1999-118173	19990913 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
AU 2000068430	A5	20010417	AU 2000-68430	20000907 <--
BR 2000013958	A	20020514	BR 2000-13958	20000907 <--
EP 1216041	A2	20020626	EP 2000-956522	20000907 <--
EP 1216041	B1	20040204		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
AT 258792	E	20040215	AT 2000-956522	20000907 <--
PRAI EP 1999-118173	A	19990913	<--	
WO 2000-EP8731	W	20000907	<--	

CLASS

PATENT NO. CLASS PATENT FAMILY CLASSIFICATION CODES

WO 2001019356 ICM A61K031-00

AB A composition for use as a medicament, functional food, or nutritional product is described which comprises at least one **lipid** which provides > 35% total energy of the composition. A preferred embodiment comprises an **n-6/n-3 fatty acid** ratio of about 2:1 to 7:1. In addition, a method of preparing the composition, use of the composition in the manufacture of a medicament or nutritional product, and a method of treatment or prevention of **sepsis** or inflammatory shock comprising administering an effective amount of the composition are described. An example showing that a high **lipid** diet (15% and 35% **lipids**) limits body weight loss in a rat model of **sepsis** was presented. A high-lipid diet had a beneficial effect for limitation of N loss induced by **sepsis**, suggesting a potential decrease of muscle proteolysis (which is dramatically increased in acute inflammatory conditions). It was particularly effective if the diet has been enriched with **lipids** before infection.

ST **lipid omega fatty acid** food nutrient;  
**sepsis** inflammatory shock **lipid** diet

IT Anti-inflammatory agents  
Drug delivery systems  
Food  
Nutrients  
Sepsis  
(compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT Canola oil  
**Fatty acids**, biological studies  
Olive oil  
Safflower oil  
Soybean oil  
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fats and Glyceridic oils**, biological studies  
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(fish; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Lipids**, biological studies  
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Diet**  
(high-lipid; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

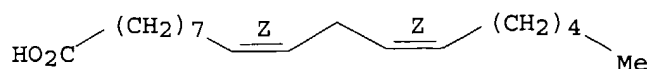
IT **Shock (circulatory collapse)**  
(inflammatory; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Glycerides**, biological studies  
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(medium-chain; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

IT **Fats and Glyceridic oils**, biological studies  
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(milk; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)

- IT **Fatty acids, biological studies**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (monounsatd.; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT **Fatty acids, biological studies**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (polyunsatd., n-3; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT **Fatty acids, biological studies**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (polyunsatd., omega-6; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT **Fatty acids, biological studies**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (polyunsatd.; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT **Fatty acids, biological studies**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (saturated; compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 60-33-3, **Linoleic acid**, biological studies 112-80-1, 9-Octadecenoic acid (9Z)-, biological studies 463-40-1,  $\alpha$ -**Linolenic acid** 506-26-3,  $\gamma$ -**Linolenic acid** 506-32-1, **Arachidonic acid** 544-63-8, Tetradecanoic acid, biological studies 6217-54-5, DHA 10417-94-4, **Eicosapentaenoic acid** 32839-34-2, **Docosapentaenoic acid** 92661-11-5, Dihomo- $\gamma$ -**linoleinic acid**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- IT 60-33-3, **Linoleic acid**, biological studies 463-40-1,  $\alpha$ -**Linolenic acid** 506-26-3,  $\gamma$ -**Linolenic acid** 506-32-1, **Arachidonic acid** 6217-54-5, DHA 10417-94-4, **Eicosapentaenoic acid** 32839-34-2, **Docosapentaenoic acid** 92661-11-5, Dihomo- $\gamma$ -**linoleinic acid**  
 RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (compns. of high-lipid diet for prevention or treatment of **sepsis** or inflammatory shock)
- RN 60-33-3 HCAPLUS  
 CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

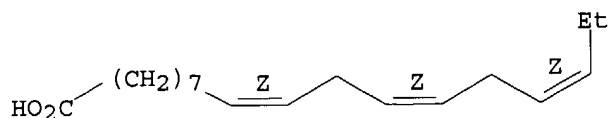
Double bond geometry as shown.





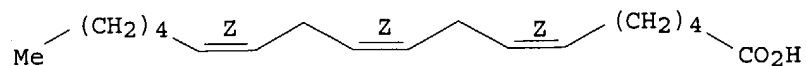
RN 463-40-1 HCAPLUS  
 CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



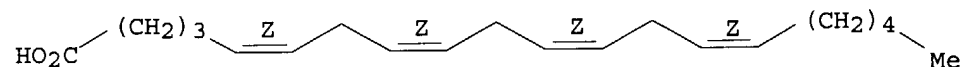
RN 506-26-3 HCAPLUS  
 CN 6,9,12-Octadecatrienoic acid, (6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



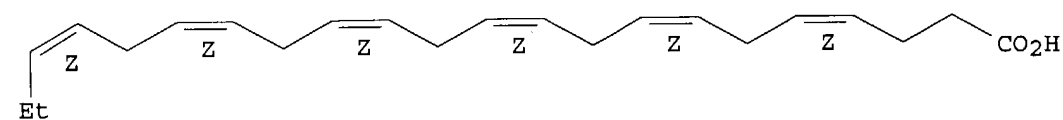
RN 506-32-1 HCAPLUS  
 CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



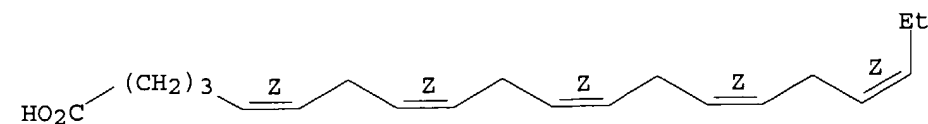
RN 6217-54-5 HCAPLUS  
 CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS  
 CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 32839-34-2 HCAPLUS  
 CN Docosapentaenoic acid, (Z,Z,Z,Z,Z)- (9CI) (CA INDEX NAME)

CM 1

CRN 112-85-6

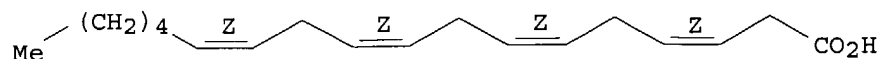
CMF C22 H44 O2



RN 92661-11-5 HCAPLUS

CN 3,6,9,12-Octadecatetraenoic acid, (3Z,6Z,9Z,12Z) - (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 2000:237890 HCAPLUS

DN 133:104372

ED Entered STN: 13 Apr 2000

TI Effects of parenteral infusion with fish-oil or safflower-oil emulsion on hepatic **lipids**, plasma amino acids, and inflammatory mediators in **septic rats**

AU Chao, C. Y.; Yeh, S. L.; Lin, M. T.; Chen, W. J.

CS Institute of Nutrition and Health Science, Taipei Medical College, Taipei, Taiwan

SO Nutrition (New York) (2000), 16(4), 284-288

CODEN: NUTRER; ISSN: 0899-9007

PB Elsevier Science Inc.

DT Journal

LA English

CC 18-5 (Animal **Nutrition**)

AB This study was designed to investigate the effects of preinfusion with total parenteral nutrition (TPN) using fish-oil (FO) vs. safflower-oil (SO) emulsion as fat sources on hepatic **lipids**, plasma amino-acid profiles, and inflammatory-related mediators in **septic rats**. Normal rats, with internal jugular catheters, were assigned to two different groups and received TPN. TPN provided 300 kcal · kg<sup>-1</sup> · d<sup>-1</sup>, with 40% of the non-protein energy as fat. All TPN solns. were isonitrogenous and identical in nutrient composition except for the fat emulsion, which was made of SO or FO. After receiving TPN for 6 d, each group of rats was further divided into control and **sepsis** subgroups. **Sepsis** was induced by cecal ligation and puncture; control rats received sham operation. All rats were classified into four groups as follows: FO control group (FOC; n = 7), FO **sepsis** group (FOS; n = 8), SO control group (SOC; n = 8), and SO **sepsis** group (SOS; n = 9). The results of the study demonstrated that plasma concns. of triacylglycerol and non-esterified **fatty acids** did not differ between the FO and SO groups, regardless of whether the animals were **septic**. SOS had significantly higher total **lipids** and cholesterol content in the liver than did the SOC group. The FOS group, however, showed no difference from the FOC group. Plasma leucine and isoleucine levels were significantly lower in the SOS group than in the SOC group, whereas no difference in these two amino acids was observed between the FOC and FOS groups. Plasma arginine levels were significantly lower in both **septic** groups than in the groups without **sepsis** when either FO or SO was infused. Plasma glutamine levels, however, did not differ across groups. No differences in interleukin-1β, interleukin-6, tumor necrosis factor-α, or leukotriene B4 concns. in peritoneal lavage fluid were observed between the two **septic** groups. These results suggest that catabolic reaction in **septic rats** preinfused with FO is not as obvious as those preinfused with SO. Compared with SO emulsion, TPN with FO emulsion prevents liver fat accumulation associated with

- sepsis.** However, parenterally administered FO had no beneficial effect in lowering cytokines and LTB4 levels in peritoneal lavage fluid in **septic rats** induced by cecal ligation and puncture.
- ST fish oil liver **lipid** amino acid; safflower oil liver **lipid** amino acid; inflammation cytokines immunity fish safflower oil
- IT **Fats and Glyceridic oils, biological studies**  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)  
 (fish; parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT Blood plasma  
 Inflammation  
 Liver  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT Safflower oil  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT Amino acids, biological studies  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT Cytokines  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT **Glycerides**, biological studies  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT Interleukins  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT **Lipids**, biological studies  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 (parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT **Nutrition**, animal  
 (parenteral; parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic **lipids**, plasma amino acids, and inflammatory mediators)
- IT 56-86-0, Glutamic acid, biological studies 57-88-5, Cholesterol, biological studies 61-90-5, Leucine, biological studies 73-32-5, Isoleucine, biological studies 74-79-3, Arginine, biological studies  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(parenteral infusion of fish-oil or safflower-oil emulsion effect on hepatic lipids, plasma amino acids, and inflammatory mediators)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD  
RE

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L78 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 1998:258015 HCAPLUS

DN 129:52707

ED Entered STN: 07 May 1998

TI Lipid mediators in inflammatory disorders

AU Heller, Axel; Koch, Thea; Schmeck, Joachim; Van Ackern, Klaus

CS Department of Anaesthesiology and Intensive Care Medicine, University of Dresden, Dresden, Germany

SO Drugs (1998), 55(4), 487-496  
CODEN: DRUGAY; ISSN: 0012-6667

PB Adis International Ltd.

DT Journal; General Review

LA English

CC 14-0 (Mammalian Pathological Biochemistry)  
Section cross-reference(s): 2

- AB A review, with 46 refs. During the past few decades, intensive collaborative research in the fields of chronic and acute inflammatory disorders has resulted in a better understanding of the pathophysiol. and diagnosis of these diseases. Modern therapeutic approaches are still not satisfactory and shock, **sepsis** and multiple organ failure remain the great challenge in intensive care medicine. However, the treatment of inflammatory diseases like rheumatoid arthritis, ulcerative colitis or psoriasis also represents an unresolved problem. Many factors contribute to the complex course of inflammatory reactions. Microbiol., immunol. and toxic agents can initiate the inflammatory response by activating a variety of humoral and cellular mediators. In the early phase of inflammation, excessive amts. of interleukins and **lipid** -mediators are released and play a crucial role in the pathogenesis of organ dysfunction. **Arachidonic acid** (AA), the mother substance of the pro-inflammatory eicosanoids, is released from membrane **phospholipids** in the course of inflammatory activation and is metabolized to prostaglandins and leukotrienes. Various strategies have been evaluated to control the excessive production of **lipid** mediators on different levels of biochem. pathways, such as inhibition of phospholipase A2, the trigger enzyme for release of AA, blockade of cyclo-oxygenase and lipoxygenase pathways and the development of receptor antagonists against platelet activating factor and leukotrienes. Some of these agents exert protective effects in different inflammatory disorders such as **septic** organ failure, rheumatoid arthritis or asthma, whereas others fail to do so. Encouraging results have been obtained by dietary supplementation with long chain  $\omega$ -3 **fatty acids** like **eicosapentaenoic acid** (EPA). In states of inflammation, EPA is released to compete with AA for enzymic metabolism inducing the production of less inflammatory and chemotactic derivs.
- ST **lipid** mediator inflammatory disease review
- IT Disease, animal  
Inflammation  
(**lipid** mediators in inflammatory disorders)
- IT Cytokines  
Eicosanoids  
**Lipids**, biological studies  
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)  
(**lipid** mediators in inflammatory disorders)
- RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
- RE
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L78 ANSWER 4 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 1997:505727 HCAPLUS

DN 127:126653

ED Entered STN: 11 Aug 1997

TI Pharmaceutical composition comprising L-carnitine or an alkanoyl L-carnitine in combination with a **polyunsaturated fatty acid** of the **omega-3** series for the prevention and the treatment of **lipid** metabolism disorders

IN Calvani, Menotti; Cavazza, Claudio

PA Sigma-Tau Industrie Farmaceutiche Riunite S.P.A., Italy

SO Eur. Pat. Appl., 7 pp.

CODEN: EPXXDW

DT Patent

LA English

IC ICM A61K031-205

ICI A61K031-205, A61K031-20

CC 63-6 (Pharmaceuticals)

Section cross-reference(s): 1

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 780124	A1	19970625	EP 1996-118681	19961121 <--
	EP 780124	B1	20011219		
	R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	AT 210977	E	20020115	AT 1996-118681	19961121 <--
	PT 780124	T	20020628	PT 1996-118681	19961121 <--
	ES 2169197	T3	20020701	ES 1996-118681	19961121 <--
	US 5753703	A	19980519	US 1996-755310	19961122 <--
	TW 522013	B	20030301	TW 1996-85114597	19961126 <--
	CA 2191645	AA	19970622	CA 1996-2191645	19961129 <--
	JP 09176005	A2	19970708	JP 1996-335456	19961216 <--
	ZA 9610769	A	19970709	ZA 1996-10769	19961220 <--
PRAI	IT 1995-RM835	A	19951221	<--	

## CLASS

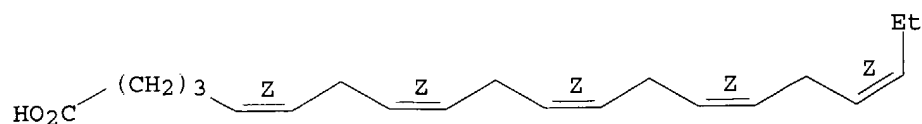
PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES	
EP 780124	ICM	A61K031-205	
	ICI	A61K031-205, A61K031-20	
US 5753703	ECLA	A61K031/205; A61K031/22F	<--
AB	A novel therapeutic use of L-carnitine (I) or alkanoyl L-carnitine is disclosed in combination with a <b>polyunsatd. fatty acid</b> of the <b>omega-3</b> series, for the prevention and the treatment of <b>lipid</b> metabolism disorders and cardiovascular disorders. Rats with exptl. induced atherosclerotic lesions were treated with 100 mg I/kg and 2 mL fish oil/kg (containing 18% <b>eicosapentaenoic acid</b> and 12% <b>docosahexaenoic acid</b> ) for 6 wk. No lesions were detectable in the treated group, revealing a surprising degree of synergism between I and <b>polyunsatd. fatty acids</b> of the <b>omega-3</b> series. A pharmaceutical composition contained I 250, <b>polyunsatd. fatty acids</b> of fish 1 (containing 350 mg <b>eicosapentaenoic acid</b> and 150 mg <b>docosahexaenoic acid</b> ), and $\alpha$ -tocopherol acetate 1 mg.		
ST	pharmaceutical carnitine <b>polyunsatd omega fatty acid</b> ; <b>lipid</b> metab disorder <b>omega fatty acid</b> ; fish oil <b>eicosapentaenoic acid</b> antiatherosclerotic; <b>docosahexaenoic acid</b> fish oil antiatherosclerotic		
IT	Antiartherosclerotics (antiatherosclerotics; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	Drug delivery systems (capsules; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	<b>Fats and Glyceridic oils, biological studies</b> RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (fish; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	Drug delivery systems (granules; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	Drug delivery systems (liposomes; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	Drug delivery systems (liqs.; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention and treatment of <b>lipid</b> metabolism disorders)		
IT	<b>Lipids, biological studies</b> RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (metabolism, disorders; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with <b>polyunsatd. omega-3 fatty acid</b> for prevention		

- and treatment of **lipid** metabolism disorders)
- IT Salts, biological studies  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (mineral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Nerve, disease  
 (neuropathy, diabetic, peripheral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
 (oral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
 (parenterals; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Nerve, disease  
 (peripheral, diabetic neuropathy; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Blood vessel, disease  
 (peripheral; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT **Anaphylaxis**  
 Anti-inflammatory agents  
 Anticoagulants  
 Antioxidants  
 Cardiovascular agents  
 Drug delivery systems  
**Hypertriglyceridemia**  
 Propolis  
**Shock (circulatory collapse)**  
 (pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Corn oil  
 Vitamins  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT **Fatty acids, biological studies**  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (**polyunsatd., n-3**; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid**)



- acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
(powders; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
(rectal; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT **Shock (circulatory collapse)**  
(**septic**; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
(solids; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT Drug delivery systems  
(tablets; pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT 112-85-6, Behenic acid 541-15-1, L-Carnitine 3040-38-8, Acetyl L-carnitine **10417-94-4, Eicosapentaenoic acid**  
20064-19-1, Propionyl L-carnitine 25576-40-3, Butyryl L-carnitine 31023-24-2, Isovaleryl L-carnitine 40225-14-7, Valeryl L-carnitine 81926-94-5 86227-47-6  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)  
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- IT **10417-94-4, Eicosapentaenoic acid**  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)  
(pharmaceutical composition comprising carnitine or alkanoyl carnitine in combination with **polyunsatd. omega-3 fatty acid** for prevention and treatment of **lipid** metabolism disorders)
- RN 10417-94-4 HCAPLUS
- CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



ED Entered STN: 20 Aug 1996  
 TI Bioactive agent-containing biocomplex for correcting biological  
 information transfer using three biological information blocks  
 IN Danielov, Michael M.  
 PA Dns Scientific, Inc., USA  
 SO PCT Int. Appl., 149 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 IC ICM A61K038-21  
 ICS A61K039-395; A61K031-55; A61K031-44; A61K031-24  
 CC 1-12 (Pharmacology)  
 Section cross-reference(s): 2, 62, 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9617621	A1	19960613	WO 1995-US15919	19951206 <--
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	US 5885974	A	19990323	US 1994-350234	19941206 <--
	AU 9645108	A1	19960626	AU 1996-45108	19951206 <--
	US 6303588	B1	20011016	US 1999-228384	19990112 <--
PRAI	US 1994-350234	A	19941206	<--	
	WO 1995-US15919	W	19951206	<--	

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 9617621	ICM	A61K038-21
	ICS	A61K039-395; A61K031-55; A61K031-44; A61K031-24
WO 9617621	ECLA	A61K007/06C4F; A61K008/60C; A61K008/63; A61K008/64; A61K008/67; A61K008/67F; A61K008/67F3; A61K008/67L; A61K038/22; A61K038/33; A61K038/35; A61K038/39; A61K038/45; A61K038/46; A61K038/4; A61K045/06; A61K045/06; A61Q019/00; A61K007/48C4F; A61K007/48C18; A61K008/06C; A61K008/365; A61K; A61K008/55C <--
US 5885974	ECLA	A61K007/06C4F; A61K007/06C18; A61K007/06C16M; A61K007/06C20; A61K007/48C4F; A61K007/48C18; A61K007/48C16D; A61K007/48C22; A61K009/127M; A61K038/22; A61K038/33; A61K038/35; A61K038/39; A61K038/45; A61K008/46; A61K038/48; A61K045/06; A61K045/06 <--

AB Methods are disclosed for correcting biol. information transfer in a patient in need of such therapy which comprise administration of a composition comprising a therapeutically effective amount of a biocomplex comprising  $\geq 1$  bioactive agent from each of the 3 informational blocks of biol. information transfer, each agent present in an amount sufficient to correct the biol. information transfer of the patient under treatment and resulting in the resumption of normal cell metabolism, and the amount being

less

than the buffering amount of said agent; together with a carrier therefor.

ST biol information transfer block therapeutic; cell metab information transfer biocomplex therapeutic

IT Acne  
 Alopecia  
 Animal cell  
 Antioxidants  
 Circulation  
 Cosmetics

Eczema

Metabolism

**Pharmaceutical** dosage forms

**Pharmaceuticals**

Pruritus

Psoriasis

Seborrhea

Signal transduction, biological

Skin, disease

Therapeutics

(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Albumins, biological studies

Calmodulins

Carbohydrates and Sugars, biological studies

Catecholamines

Cerebrosides

Coenzymes

Collagens, biological studies

Elastins

Gelatins, biological studies

**Glycolipids**

**Lipids**, biological studies

Orosomucoids

Peptides, biological studies

Phosphatidic acids

Phosphatidylcholines, biological studies

Phosphatidylethanolamines

Phosphatidylinositols

Phosphatidylserines

Phosphoinositides

**Phospholipids**, biological studies

Prostaglandins

Protamines

Proteins, biological studies

**Sphingolipids**

Steroids, biological studies

Sulfatides

Vitamins

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Animal growth regulator receptors

Estrogen receptors

Prostaglandin receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Brain

(extract; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT **Shock**

(post-trauma; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Cell membrane

(substitute cell membrane delivery system; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT Prostaglandins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL

- (Biological study); PROC (Process)  
(A, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Prostaglandins  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(D, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Prostaglandins  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(E, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Receptors  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(animal growth regulator, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Skin  
(cellulite, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT **Glycerides**  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(di-, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Phosphoinositides  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(di-, 4-phosphates, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Skin, disease  
(dry, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Receptors  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(estrogen, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Corticosteroid receptors  
Receptors  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
(glucocorticosteroid, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Lipoproteins  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(high-d., bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Phosphatidylcholines, biological studies  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
(hydrogenated, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Elastins  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic

- use); BIOL (Biological study); USES (Uses)  
 (hydrolyzates, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Lipoproteins  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological  
 study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic  
 use); BIOL (Biological study); USES (Uses)  
 (low-d., bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Corticosteroid receptors  
 Receptors  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
 (Biological study); PROC (Process)  
 (mineralocorticosteroid, bioactive agent-containing biocomplex for  
 correcting biol. information transfer and cell metabolism, and therapeutic  
 use)
- IT Dermatitis  
 (neuro-, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Skin, disease  
 (oily, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT **Pharmaceutical** dosage forms  
 (ointments, creams, bioactive agent-containing biocomplex for correcting  
 biol. information transfer and cell metabolism, and therapeutic use)
- IT **Pharmaceutical** dosage forms  
 (ophthalmic, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT **Pharmaceutical** dosage forms  
 (parenterals, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Receptors  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
 (Biological study); PROC (Process)  
 (prostaglandin, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Sunburn and Suntan  
 (suntanning agents, bioactive agent-containing biocomplex for correcting  
 biol. information transfer and cell metabolism, and therapeutic use)
- IT **Pharmaceutical** dosage forms  
 (topical, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Injury  
 (trauma, shock following; bioactive agent-containing biocomplex for  
 correcting biol. information transfer and cell metabolism, and therapeutic  
 use)
- IT Phosphoinositides  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological  
 study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic  
 use); BIOL (Biological study); USES (Uses)  
 (tri-, 4,5-bis(phosphates), bioactive agent-containing biocomplex for  
 correcting biol. information transfer and cell metabolism, and therapeutic  
 use)
- IT Collagens, biological studies  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological  
 study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic  
 use); BIOL (Biological study); USES (Uses)  
 (type I, bioactive agent-containing biocomplex for correcting biol.  
 information transfer and cell metabolism, and therapeutic use)
- IT Collagens, biological studies  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological  
 study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic  
 use); BIOL (Biological study); USES (Uses)

- (type II, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Collagens, biological studies  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (type III, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Lipoproteins  
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)  
 (very-low-d., bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Skin, disease  
 (wrinkle, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Receptors  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 ( $\alpha$ 2-adrenergic, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT Receptors  
 RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)  
 ( $\beta$ 2-adrenergic, bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT 60-92-4, Cyclic AMP  
 RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)  
 (bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)
- IT 50-14-6, Ergocalciferol 50-23-7, Hydrocortisone 50-28-2,  $\beta$ -Estradiol, biological studies 50-81-7, L-Ascorbic acid, biological studies 51-61-6, Dopamine, biological studies 52-39-1, Aldosterone 52-89-1, L-Cysteine hydrochloride 53-59-8,  $\beta$ -NADP 53-84-9,  $\beta$ -NAD 54-47-7, Pyridoxal-5-phosphate 55-31-2, Epinephrine hydrochloride 56-65-5, Adenosine triphosphate, biological studies 56-81-5D, 1,2,3-Propanetriol, 1,2-diacyl derivs. 56-89-3, L-Cystine, biological studies 57-11-4, Octadecanoic acid, biological studies 57-83-0, Progesterone, biological studies 57-87-4, Ergosterol 57-88-5, Cholesterol, biological studies 58-56-0, Pyridoxine hydrochloride 58-85-5, Biotin 58-95-7,  $\alpha$ -Tocopherol acetate 59-30-3, Folic acid, biological studies 60-18-4, L-Tyrosine, biological studies 60-33-3, 9,12-Octadecadienoic acid (Z,Z)-, biological studies 63-91-2, L-Phenylalanine, biological studies 65-71-4, Thymine 66-22-8, Uracil, biological studies 67-03-8, Thiamine hydrochloride 71-30-7, Cytosine 73-22-3, L-Tryptophan, biological studies 73-24-5, Adenine, biological studies 73-40-5, Guanine 79-81-2, Retinol palmitate 85-61-0, Coenzyme A, biological studies 86-01-1, Guanosine triphosphate 96-26-4, Dihydroxyacetone 98-92-0, Nicotinamide 112-85-6, Behenic acid 113-79-1, Arginine vasopressin 117-39-5, Quercetin 122-32-7, Triolein 123-33-1, Maleic hydrazide 135-16-0, Tetrahydrofolic acid 137-08-6, Pantothenic acid hemicalcium salt 145-42-6, Sodium taurocholate 154-87-0, Cocarboxylase 329-56-6, Arterenol hydrochloride 361-09-1, Sodium cholate 363-24-6, Prostaglandin E2 463-40-1, Linolenic acid 481-39-0, Juglone 506-21-8, Linolelaidic acid 506-30-9, Arachidic acid 537-40-6, Trilinolein 551-11-1, Prostaglandin F2 $\alpha$  555-43-1, Tristearin 606-68-8 620-64-4, Triarachidin 745-65-3, Prostaglandin E1 863-57-0, Sodium glycocholate 987-65-5, Adenosine triphosphate disodium salt 1105-02-8, Corticosterone-21-sulfate 1184-16-3

1340-08-5, Vitamin P 1407-47-2, Angiotensin 1731-94-8, Nonadecanoic acid methyl ester 2566-90-7 2644-64-6, Dipalmitoylphosphatidylcholine 2752-99-0, Trierucin 4537-76-2, Distearoylphosphatidylethanolamine 4537-77-3, Dipalmitoylphosphatidylglycerol 4537-78-4, Distearoylphosphatidylglycerol 4539-70-2, Distearoylphosphatidylcholine 4999-79-5, Estradiol-3-sulfate sodium salt 5681-36-7, Dipalmitoylphosphatidylethanolamine 6064-90-0, Heneicosanoic acid methyl ester 6610-25-9, **Arachidonic acid** sodium salt 7235-40-7,  $\beta$ -Carotene 7665-99-8, Cyclic GMP 9001-62-1, Lipase 9002-60-2, Adrenocorticotrophic hormone, biological studies 9002-60-2D, Adrenocorticotrophic hormone, 1-24 fragment 9002-64-6, Parathyroid hormone 9002-64-6D, Parathyroid hormone, 1-36 fragment 9002-67-9, Luteinizing hormone 9002-68-0, Follicle-stimulating hormone 9002-71-5, Thyrotrophic hormone 9002-72-6, Somatotropin 9004-10-8, Insulin, biological studies 9004-61-9, Hyaluronic acid 9005-49-6, Heparin sulfate, biological studies 9007-12-9, Thyrocalcitonin 9007-92-5, Glucagon, biological studies 9015-73-0 9026-43-1, Protein kinase 9041-08-1, Heparin sodium salt **10417-94-4** 10529-43-8, Cholecalciferol sulfate 11000-17-2, Vasopressin 11061-68-0, Human insulin 11128-99-7, Angiotensin II 12629-01-5, Human growth hormone 13487-42-8 14465-68-0 15866-84-9, Adenosine triphosphate calcium salt 18641-57-1, Trihehenin 18656-38-7, Dimyristoylphosphatidylcholine 20255-95-2, Dimyristoylphosphatidylethanolamine 20290-75-9 22251-85-0, Flavin mononucleotide sodium salt 24967-93-9, Chondroitin sulfate A 24967-94-0, Dermatan sulfate 25322-46-7, Chondroitin sulfate C 26536-13-0, Trinadecanoic acid 27964-99-4, Poly-D-lysine hydrobromide 28845-86-5, 13,16,19-Docosatrienoic acid, (Z,Z,Z)- 28874-58-0 35121-78-9, Prostaglandin I<sub>2</sub> 37221-79-7, Vasoactive intestinal peptide 37377-93-8,  $\beta$ -Lipotropin 37377-93-8D,  $\beta$ -Lipotropin, fragment 37839-81-9, Cyclic AMP sodium salt 40245-60-1, Cyclic GMP sodium salt 41598-07-6, Prostaglandin D<sub>2</sub> 52910-82-4, Aldosterone-21-hemisuccinate 55672-92-9, Coenzyme A sodium salt 59392-49-3, Gastric inhibitory peptide 60617-12-1,  $\beta$ -Endorphin 60617-12-1D,  $\beta$ -Endorphin, fragment 61361-72-6, Dimyristoylphosphatidylglycerol 61849-14-7, Prostaglandin I<sub>2</sub> sodium salt 78392-27-5, Cholecalciferol sulfate sodium salt 80380-39-8, Tri-11-eicosenoic acid 85166-31-0, D-myo-Inositol-1,4,5-triphosphate 92216-45-0, D-myo-Inositol-2,4,5-triphosphate 96012-99-6, Guanosine triphosphate lithium salt 99660-95-4 100775-23-3, Corticosterone-21-sulfate potassium salt 108340-81-4, D-myo-Inositol, 1,4,5-tris(dihydrogen phosphate), hexasodium salt 135271-36-2, D-myo-Inositol-1,4,5-triphosphate potassium salt

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); **THU** (Therapeutic use); BIOL (Biological study); USES (Uses)

(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT 7440-70-2, Calcium, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(intracellular, mobilization; bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

IT 60-33-3, 9,12-Octadecadienoic acid (Z,Z)-, biological studies

**463-40-1, Linolenic acid 10417-94-4**

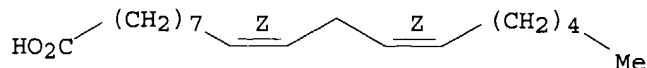
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BUU (Biological use, unclassified); **THU** (Therapeutic use); BIOL (Biological study); USES (Uses)

(bioactive agent-containing biocomplex for correcting biol. information transfer and cell metabolism, and therapeutic use)

RN 60-33-3 HCAPLUS

CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

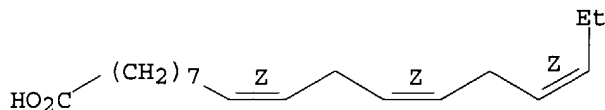
Double bond geometry as shown.



RN 463-40-1 HCAPLUS

CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

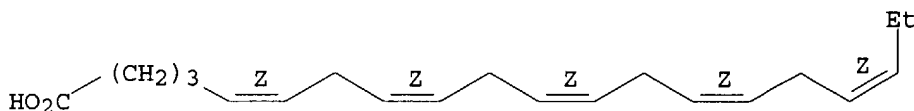
Double bond geometry as shown.



RN 10417-94-4 HCAPLUS

CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 6 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 1994:186801 HCAPLUS

DN 120:186801

ED Entered STN: 16 Apr 1994

TI **Lipid** microemulsions for culture media

IN Inlow, Duane

PA USA

SO Can., 35 pp.

CODEN: CAXXA4

DT Patent

LA English

IC ICM C12N005-00

CC 9-11 (Biochemical Methods)

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CA 1315725	A1	19930406	CA 1988-572547	19880720 <--
	EP 377582	A1	19900718	EP 1988-906679	19880720 <--
	EP 377582	B1	19971015		
	R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
	JP 02504225	T2	19901206	JP 1988-506722	19880720 <--
	JP 2788270	B2	19980820		
	AT 159287	E	19971115	AT 1988-906679	19880720 <--
	IL 87195	A1	19940412	IL 1988-87195	19880722 <--
	AU 8943124	A1	19900418	AU 1989-43124	19890913 <--
	US 5372943	A	19941213	US 1993-90568	19930712 <--
PRAI	US 1987-77189	A	19870724	<--	
	WO 1988-US2440		19880720	<--	
	US 1988-248830	A	19880923	<--	
	WO 1989-US3985	A	19890913	<--	
	US 1992-829610	B1	19920130	<--	

CLASS

PATENT NO. CLASS PATENT FAMILY CLASSIFICATION CODES



$$\text{HO}(\text{CH}_2\text{CH}_2\text{O})_t - \text{C}_6\text{H}_2(\text{OCH}_2\text{CH}_2)_u\text{OH} - \text{CH}_2\text{CO}_2\text{R}$$

AB **Lipids** are supplied in cell culture media in the form of a microemulsion comprising (1) an aqueous component containing  $\geq 1$  emulsifier in water, said emulsifier being selected from **phospholipids** and nontoxic, nonionic, polymeric detergents, and (2) a **lipid** component containing  $\geq 1$  **lipid** and  $\geq 1$  emulsifier in an organic solvent. The detergent emulsifiers are either (1) block copolymers of propylene oxide and ethylene oxide or (2) polysorbates I (R = (un)saturated C16-20 **fatty acid**; t = 10-30; u = 10-20). Thus, a solution of cod liver oil 100, Tween 80 250, cholesterol 45, and  $\alpha$ -tocopherol acetate 20 mg in 10 mL EtOH was filter sterilized and 1 mL was mixed **aseptically** with 10 mL Pluronic F68 for addition to 1 L culture medium for insect cells. *Spodoptera frugiperda* cells grown in this medium showed a growth rate, maximum cell d., and cell viability comparable to those of cells grown in medium containing 10% serum.

ST **lipid** microemulsion cell culture medium

IT **Phospholipids**, biological studies  
RL: BIOL (Biological study)  
(as emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)

IT Lecithins  
Polymers, biological studies  
RL: BIOL (Biological study)  
(as nonionic emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)

IT Emulsifying agents  
(for **lipid** microemulsions, in serum-free cell culture media)

IT Antioxidants  
Reducing agents  
Cod-liver oil  
RL: BIOL (Biological study)  
(**lipid** microemulsions containing, in serum-free cell culture media)

IT Animal tissue culture  
(**lipid** microemulsions in serum-free culture media for)

IT **Lipids**, biological studies  
RL: BIOL (Biological study)  
(microemulsions, in serum-free cell culture media)

IT **Fatty acids**, esters  
RL: BIOL (Biological study)  
(Me esters, microemulsions, in serum-free cell culture media)

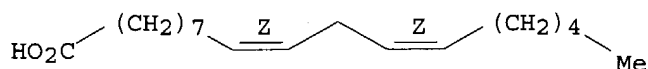
IT Animal cell line  
(SF9, **lipid** microemulsions in serum-free culture media for)

IT Vitamins  
RL: BIOL (Biological study)  
(fat-soluble, microemulsions, in serum-free cell culture media)

IT **Fats and Glyceridic oils**  
RL: BIOL (Biological study)  
(**fish**-liver, microemulsions, in serum-free cell culture

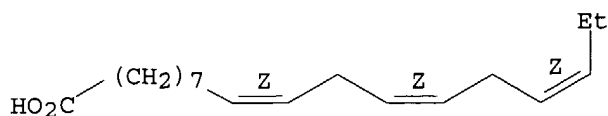
- media)
- IT Steroids, biological studies  
RL: BIOL (Biological study)  
(hydroxy, microemulsions, in serum-free cell culture media)
- IT Emulsions  
(micro-, **lipid**, in serum-free cell culture media)
- IT Detergents  
(nonionic, polymers, as emulsifying agents in **lipid** microemulsions for serum-free cell culture media)
- IT **Fatty acids**, biological studies  
RL: BIOL (Biological study)  
(**unsatd.**, microemulsions, in serum-free cell culture media)
- IT 9005-65-6, Polyoxyethylene sorbitan monooleate 106392-12-5, Ethylene oxide-propylene oxide block copolymer  
RL: BIOL (Biological study)  
(as nonionic emulsifying agent, in **lipid** microemulsions for serum-free cell culture media)
- IT 9005-63-4D, Sorbitan poly(oxy-1,2-ethanediyl) derivs., fatty esters  
RL: BIOL (Biological study)  
(as nonionic emulsifying agents, in **lipid** microemulsions for serum-free cell culture media)
- IT 38098-46-3, Monothioglycerol  
RL: BIOL (Biological study)  
(**lipid** microemulsions containing, as antioxidant in serum-free cell culture media)
- IT 58-95-7,  $\alpha$ -Tocopherol acetate  
RL: BIOL (Biological study)  
(**lipid** microemulsions containing, in serum-free cell culture media)
- IT 57-88-5, Cholesterol, biological studies **60-33-3**, **Linoleic acid**, biological studies 112-80-1, Oleic acid, biological studies **463-40-1**, **Linolenic acid** 1406-16-2, Vitamin D 1406-18-4, Vitamin E 11103-57-4, Vitamin A  
RL: BIOL (Biological study)  
(microemulsions, in serum-free cell culture media)
- IT **60-33-3**, **Linoleic acid**, biological studies **463-40-1**, **Linolenic acid**  
RL: BIOL (Biological study)  
(microemulsions, in serum-free cell culture media)
- RN 60-33-3 HCAPLUS  
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



- RN 463-40-1 HCAPLUS  
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



DN 118:132151  
 ED Entered STN: 30 Mar 1993  
 TI Therapeutic nutrients containing **glycerides** of branched chain  
**fatty acids**  
 IN Sommermeyer, Klaus  
 PA Fresenius A.-G., Germany  
 SO Ger. Offen., 6 pp.  
 CODEN: GWXXBX  
 DT Patent  
 LA German  
 IC ICM A23L001-29  
 ICS A23K001-00; A23K001-18; A61K031-22; A61K031-23; C07C069-30;  
 C07C067-08  
 ICA A23L001-035; A61K009-107; A61K009-48; A23L001-0562; A23P001-04  
 CC 63-6 (**Pharmaceuticals**)  
 Section cross-reference(s): 17  
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	DE 4116004	A1	19921119	DE 1991-4116004	19910516 <--
	DE 4116004	C2	19930930		
	WO 9220241	A1	19921126	WO 1992-EP1049	19920513 <--
	W: JP, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE				
	EP 584159	A1	19940302	EP 1992-910171	19920513 <--
	EP 584159	B1	19951227		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 06511384	T2	19941222	JP 1992-509184	19920513 <--
	AT 132012	E	19960115	AT 1992-910171	19920513 <--
	ES 2081108	T3	19960216	ES 1992-910171	19920513 <--
	US 5492713	A	19960220	US 1994-150039	19940805 <--
PRAI	DE 1991-4116004		19910516	<--	
	WO 1992-EP1049		19920513	<--	

## CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
DE 4116004	ICM	A23L001-29
	ICS	A23K001-00; A23K001-18; A61K031-22; A61K031-23; C07C069-30; C07C067-08
	ICA	A23L001-035; A61K009-107; A61K009-48; A23L001-0562; A23P001-04

OS MARPAT 118:132151

AB **Glycerides** containing branched **fatty acids**,  
 $\text{MeRCH}(\text{CH}_2)_m(\text{CH}_2)_n\text{CO}_2\text{H}$  (R = Me, Et; n = 0 or even number from 2-18; m = 0,1)  
 are used in therapeutic diets for treatment of trauma and **sepsis**  
 , especially uremia. These may be used in oral or parenteral formulations.  
 These **fatty acids** act as a source of branched-chain  
 keto-acids for use in branched chain amino acid biosynthesis to maintain  
 protein synthesis in the liver. The preparation of glyceryl triisovalerate,  
 glyceryl tri-(3-methylvalerate), and glyceryl tri-(4-methylvalerate)  
 by acid-catalyzed esterification is demonstrated. Formulations and dosage  
 forms (capsules) using these **glycerides** are described.

ST **glyceride** branched chain therapeutic nutrient uremia

IT Cottonseed oil

Fats and **Glyceridic** oils

Olive oil

Safflower oil

Soybean oil

Sunflower oil

RL: BIOL (Biological study)

(emulsions containing, in therapeutic nutrients for treatment of uremia)

IT **Glycerides**, biological studies

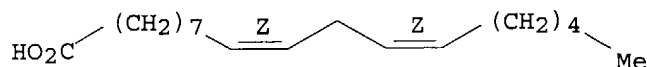
RL: BIOL (Biological study)

- (C6-24, emulsions containing, in therapeutic nutrients for treatment of uremia)
- IT Fats and **Glyceridic** oils  
RL: BIOL (Biological study)  
(animal, emulsions containing, in therapeutic nutrients for treatment of uremia)
- IT **Glycerides**, biological studies  
RL: BIOL (Biological study)  
(branched, therapeutic nutrients containing, for treatment of uremia)
- IT **Pharmaceutical** dosage forms  
(capsules, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
- IT **Pharmaceutical** dosage forms  
(emulsions, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
- IT Kidney, disease  
(failure, treatment of, **glycerides** of branched-chain **fatty acids** in nutritional formulations for)
- IT **Fats and Glyceridic** oils  
RL: BIOL (Biological study)  
(**fish**, emulsions containing, in therapeutic nutrients for treatment of uremia)
- IT **Glycerides**, biological studies  
RL: BIOL (Biological study)  
(**medium-chain**, emulsions containing, in therapeutic nutrients for treatment of uremia)
- IT **Glycerides**, biological studies  
RL: BIOL (Biological study)  
(mono-, emulsions containing, in therapeutic nutrients for treatment of uremia)
- IT **Nutrients**  
(parenteral, containing **glycerides** of branched-chain **fatty acids**, for treatment of uremia)
- IT Animal **nutrition**  
(parenteral, for treatment of uremia, **glycerides** of branched-chain **fatty acids** in)
- IT 620-63-3P 146369-67-7P 146369-68-8P  
RL: PREP (Preparation)  
(preparation of, for therapeutic nutrients for treatment of uremia)
- IT 105-43-1, 3-Methylvaleric acid 503-74-2, Isovaleric acid 646-07-1, 4-Methylvaleric acid  
RL: RCT (Reactant); RACT (Reactant or reagent)  
(reaction of, in preparation of **glycerides** for therapeutic nutrients for treatment of uremia)
- L78 ANSWER 8 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN  
AN 1993:100997 HCAPLUS  
DN 118:100997  
ED Entered STN: 19 Mar 1993  
TI Improved metabolic responses to burn injury and endotoxemia with the use of a novel **lipid** source  
AU Teo, Tiew Chong  
CS Univ. Aberdeen, Aberdeen, UK  
SO (1990) 210 pp. Avail.: Univ. Microfilms Int., Order No. BRDX94895  
From: Diss. Abstr. Int. B 1992, 52(10), 5214  
DT Dissertation  
LA English  
CC 18-5 (Animal **Nutrition**)  
AB Unavailable  
ST **lipid** enteral diet burn endotoxemia; fish oil **medium chain glyceride** burn  
IT Burn

- (enteral nutrition in, **lipid** source for)
- IT **Lipids**, biological studies  
 RL: BIOL (Biological study)  
 (for enteral nutrition in burn injury)
- IT **Sepsis and Septicemia**  
 (endotoxemia, from burn injury, **lipid** source for  
 enteral nutrition in)
- IT Animal **nutrition**  
 (enteral, in burn injury, **lipid** source for)
- IT **Fats and Glyceridic oils**  
 RL: BIOL (Biological study)  
 (fish, in enteral nutrition, in burn injury)
- IT **Glycerides, biological studies**  
 RL: BIOL (Biological study)  
 (medium-chain, in enteral nutrition, in burn  
 injury)
- L78 ANSWER 9 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN  
 AN 1990:421581 HCAPLUS  
 DN 113:21581  
 ED Entered STN: 21 Jul 1990  
 TI Sequential changes in **lipid** metabolism and the **fatty**  
**acid** profile in liver **lipids** during fasting and  
**sepsis**  
 AU Larsson-Backstroem, Carin; Arrhenius, Eva; Sagge, Kristina; Lindmark,  
 Lars; Paprocki, Jolanta; Svensson, Lennart  
 CS Dep. Exp. Biol., Kabi Nutr., Stockholm, S-112 87, Swed.  
 SO Circulatory Shock (1990), 30(4), 331-47  
 CODEN: CRSHAG; ISSN: 0092-6213  
 DT Journal  
 LA English  
 CC 14-3 (Mammalian Pathological Biochemistry)  
 AB The sequential changes in **lipid** metabolism and in the **fatty**  
**acid** profile of liver **lipids** during fasting and  
**sepsis** were studied. Liver and blood specimens were taken from  
 normally fed rats and from **nonseptically** and **septically**  
 fasted rats at 5, 24, and 48 h. **Sepsis** was induced by injecting  
 live *Escherichia coli* bacteria i.p. **Sepsis** attenuated the  
 fasting-induced increase in  $\beta$ -hydroxybutyrate and reduced liver and  
 serum **triglycerides** at 5 h. There was a percentage decline in  
 the most abundant **fatty acids** in neutral  
**lipids**, namely oleic (18:1w9) and **linoleic** (18:2w6)  
 acids. This was seen throughout fasting and **septic** fasting.  
 These results indicate that 18:1w9 and 18:2w6 are used as energy  
 substrates and are oxidized to  $\beta$ -hydroxybutyrate during fasting and  
 mainly to carbon dioxide during **septic** fasting. On the  
 contrary, the most abundant **fatty acids** in  
**phospholipids**, stearic (18:0), arachidonic (20:4w6), and  
**docosahexaenoic** (22:6w3) acids, accumulated in neutral  
**lipids** and in **phospholipids** throughout fasting.  
 However, during **sepsis** this accumulation was reduced in neutral  
**lipids** and reversed to a level below that in the fed and fasted  
 state in **phospholipids**. These results indicate that a  
 disturbance in membrane integrity and function induced by **septic**  
 fasting may have pathophysiol. consequences for **lipid** metabolism and  
 liver function during **sepsis**.  
 ST **fatty acid** liver **lipid** metab **sepsis**  
 IT **Lipids**, biological studies  
**Phospholipids**, biological studies  
 RL: BIOL (Biological study)  
 (fatty acid profile and metabolism of, sequential  
 changes in, in liver, in fasting and **sepsis**)  
 IT **Sepsis and Septicemia**

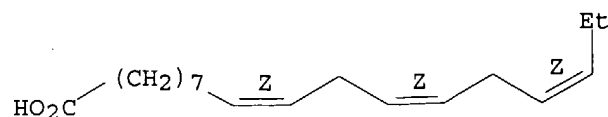
- (**lipid** metabolism by liver in fasting and, **fatty acid** profile of liver **lipids** sequential changes in relation to)
- IT Starvation  
(**lipid** metabolism by liver in **sepsis** and, **fatty acid** profile of liver **lipids** sequential changes in relation to)
- IT Liver, metabolism  
(**lipid** metabolism by, in fasting and **sepsis**, **fatty acid** profile of liver **lipids** sequential changes in)
- IT **Glycerides**, biological studies  
RL: BIOL (Biological study)  
(of **lipids** and **phospholipids** of liver, in fasting and **sepsis**)
- IT **Fatty acids**, biological studies  
RL: BIOL (Biological study)  
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)
- IT 300-85-6,  $\beta$ -Hydroxybutyric acid  
RL: BIOL (Biological study)  
(of blood serum, in fasting and **sepsis**, **lipid** metabolism by liver in relation to)
- IT 57-88-5, Cholest-5-en-3-ol (3 $\beta$ )-, biological studies  
RL: BIOL (Biological study)  
(of **lipids** and **phospholipids** of liver, in fasting and **sepsis**)
- IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 60-33-3, **Linoleic acid**, biological studies 112-80-1, 9-Octadecenoic acid (Z)-, biological studies 463-40-1 506-32-1, **Arachidonic acid** 6217-54-5 10417-94-4  
RL: BIOL (Biological study)  
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)
- IT 60-33-3, **Linoleic acid**, biological studies 463-40-1 506-32-1, **Arachidonic acid** 6217-54-5 10417-94-4  
RL: BIOL (Biological study)  
(of **lipids** and **phospholipids** of liver, sequential changes in profile of, in fasting and **sepsis**)
- RN 60-33-3 HCAPLUS  
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



- RN 463-40-1 HCAPLUS  
CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

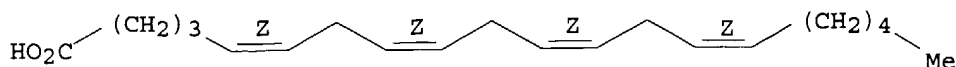
Double bond geometry as shown.



- RN 506-32-1 HCAPLUS

CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z) - (9CI) (CA INDEX NAME)

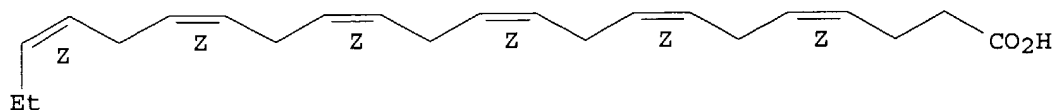
Double bond geometry as shown.



RN 6217-54-5 HCAPLUS

CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z) - (9CI) (CA INDEX NAME)

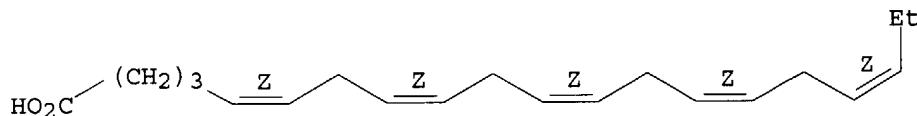
Double bond geometry as shown.



RN 10417-94-4 HCAPLUS

CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z) - (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L78 ANSWER 10 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN

AN 1988:629343 HCAPLUS

DN 109:229343

ED Entered STN: 24 Dec 1988

TI **Lipid** metabolism during starvation and **sepsis** in relation to **fatty acid** profile in liver and  $\alpha$ -**linolenic**- and  $\gamma$ -**linolenic acid**-enriched diets

AU Larsson-Backstroem, C.; Paprocki, J.; Lindmark, L.; Svensson, L.

CS Dep. Pharmacol., KabiVitrum AB, Stockholm, S-112 87, Swed.

SO NATO ASI Series, Series A: Life Sciences (1987), 139(Lipid Mediators Immunol. Shock), 125-8

CODEN: NALSDJ; ISSN: 0258-1213

DT Journal

LA English

CC 18-5 (Animal **Nutrition**)

Section cross-reference(s): 14

AB In expts. on rats (90 g), fasting for 24 h increased blood free **fatty acids** and  $\beta$ -hydroxybutyrate levels as well as the level of prekallikrein and the platelet count and decreased blood glucose. During fasting, liver **lipid** and **phospholipid** C18:2.**omega**.6 and C22:6.**omega**.3 were decreased, whereas C20:4.**omega**.6 and C22:6.**omega**.3 were accumulated in neutral **lipids**. These changes were lower during exptl. **sepsis**. The level of C20:4.**omega**.6 in **phospholipids** did not change during fasting, whereas it decreased during **sepsis**. C18:2.**omega**.6 and C20:5.**omega**.3 in **phospholipids** were reduced during fasting but unaltered in

**sepsis.** Dietary  $\alpha$ -linolenic (ALA) and  $\gamma$ -linolenic (GLA) acids increased the incorporation of  $\omega$ -3 and  $\omega$ -6 fatty acids into liver neutral lipids and phospholipids, resp., with a resulting increase in the unsatn. index. The degree of unsatn. following dietary ALA and GLA was lower during fasting and higher during **sepsis**, as compared to fed controls. Dietary ALA and GLA reduced liver triglycerides and enhanced blood free fatty acids, both in fasted and in **septic**-fasted rats. The level of  $\beta$ -hydroxybutyrate was lower during fasting, but higher during **sepsis**, compared to the control group. Evidently, there is a relation between ketogenesis and the degree of unsatn. of liver lipids.

- ST starvation lipid metab blood liver; **sepsis**  
lipid metab blood liver; linolenate diet **sepsis**  
lipid metab; fatty acid liver blood starvation  
**sepsis**
- IT Liver, composition  
(fatty acids of lipids and  
phospholipids of, starvation and **sepsis** effect on,  
dietary  $\alpha$ - and  $\gamma$ -linolenic  
acids in relation to)
- IT Phospholipids, biological studies  
RL: BIOL (Biological study)  
(fatty acids of, of liver, starvation and  
**sepsis** effect on, dietary  $\alpha$ - and  $\gamma$ -  
linolenic acids in relation to)
- IT Sepsis and Septicemia  
Starvation  
(lipid metabolism in, dietary  $\alpha$ - and  $\gamma$ -  
linolenic acids in relation to)
- IT Lipids, biological studies  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(metabolism of, starvation and **sepsis** effect on, dietary  $\alpha$ -  
and  $\gamma$ -linolenic acids effect on)
- IT Fatty acids, biological studies  
RL: BIOL (Biological study)  
(of blood and liver, starvation and **sepsis** effect on, dietary  
 $\alpha$ - and  $\gamma$ -linolenic acids in  
relation to)
- IT Glycerides, biological studies  
RL: BIOL (Biological study)  
(of liver, starvation and **sepsis** effect on, dietary  $\alpha$ -  
and  $\gamma$ -linolenic acids in relation  
to)
- IT Blood sugar  
(starvation and **sepsis** effect on, dietary  $\alpha$ - and  
 $\gamma$ -linolenic acids in relation to)
- IT Lipids, biological studies  
RL: BIOL (Biological study)  
(neutral, fatty acids of, of liver, starvation and  
**sepsis** effect on, dietary  $\alpha$ - and  $\gamma$ -  
linolenic acids in relation to)
- IT 463-40-1,  $\alpha$ -Linolenic acid  
506-26-3,  $\gamma$ -Linolenic acid  
RL: BIOL (Biological study)  
(lipid metabolism in starvation and **sepsis** in relation  
to dietary)
- IT 300-85-6 9055-02-1, Prekallikrein  
RL: BIOL (Biological study)  
(of blood, starvation and **sepsis** effect on, dietary  $\alpha$ -



and  $\gamma$  -linolenic acids in relation  
to)

IT 60-33-3, biological studies 506-32-1 6217-54-5  
10417-94-4

RL: BIOL (Biological study)  
(of neutral lipids and phospholipids, of liver,  
starvation and sepsis effect on, dietary  $\alpha$ - and  
 $\gamma$  -linolenic acids in relation to)

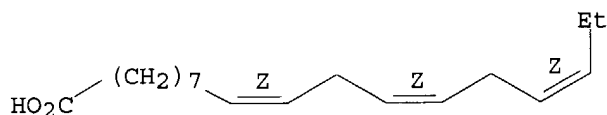
IT 463-40-1,  $\alpha$  -Linolenic acid  
506-26-3,  $\gamma$  -Linolenic acid

RL: BIOL (Biological study)  
(lipid metabolism in starvation and sepsis in relation  
to dietary)

RN 463-40-1 HCAPLUS

CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

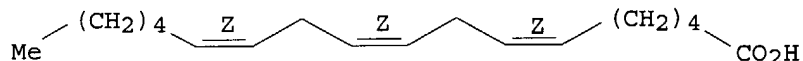
Double bond geometry as shown.



RN 506-26-3 HCAPLUS

CN 6,9,12-Octadecatrienoic acid, (6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



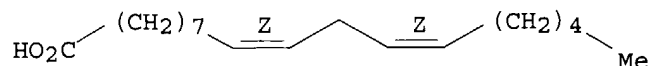
IT 60-33-3, biological studies 506-32-1 6217-54-5  
10417-94-4

RL: BIOL (Biological study)  
(of neutral lipids and phospholipids, of liver,  
starvation and sepsis effect on, dietary  $\alpha$ - and  
 $\gamma$  -linolenic acids in relation to)

RN 60-33-3 HCAPLUS

CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

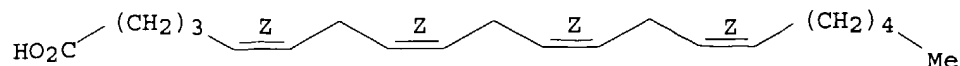
Double bond geometry as shown.



RN 506-32-1 HCAPLUS

CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 6217-54-5 HCAPLUS

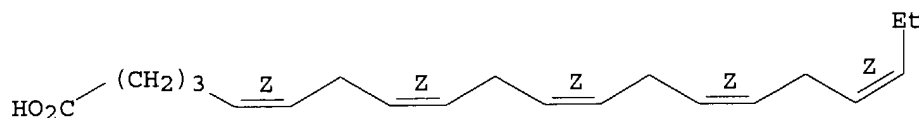
CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 10417-94-4 HCAPLUS  
 CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z) - (9CI) (CA INDEX NAME)

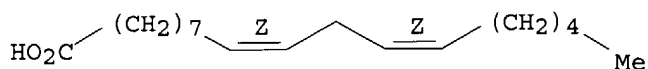
Double bond geometry as shown.



L78 ANSWER 11 OF 11 HCAPLUS COPYRIGHT 2004 ACS on STN  
 AN 1962:452201 HCAPLUS  
 DN 57:52201  
 OREF 57:10424h-i,10425a  
 ED Entered STN: 22 Apr 2001  
 TI Effects of anaphylaxis on the **lipid** metabolism of guinea pig lung  
 AU Goadby, P.; Smith, W. G.  
 CS Sunderland Tech. Coll., UK  
 SO Biochemical Journal (1962), 84, 21P  
 CODEN: BIJOAK; ISSN: 0264-6021  
 DT Journal  
 LA Unavailable  
 CC 70 (Immunochemistry)  
 AB Following anaphylaxis induced by exposing sensitized guinea pigs to aerosolized antigen, the following changes in **lipid** content of the lungs were observed: 30% loss of total cholesterol, 15% loss of total **phospholipid** during the 1st 15 min. after shock, and replacement of the lost **lipid** during the subsequent 15 min. Both neutral **lipid** and **phospholipid** fraction had normal **fatty acid** composition 15 min. after shock, but at 30 and 60 min. abnormalities were noted: less stearate and more linoleate in neutral **lipids**, less linoleate and linolenate and more palmitate in **phospholipids**. It is concluded that a loss of cholesterol and **phospholipid** (high-d. lipoprotein), stimulation of cholesterol, **glyceride**, and glycerophosphatide synthesis to replace lost constituents, and replacement of a high proportion of the lung **lipids** with newly synthesized material of abnormal **fatty acid** composition constitute the in vivo changes in **lipid** metabolism. It is suggested the distorted **lipid** metabolism may exert a profound influence on the course of disease processes having an immunological basis.  
 IT **Phospholipids**  
 (in lungs, in anaphylaxis)  
 IT Antibodies  
 (in ragweed allergy)  
 IT Lungs  
 (**lipid** metabolism by, in anaphylaxis)  
 IT **Anaphylaxis**  
 (**lipids** in lungs in, metabolism of)  
 IT **Lipids**

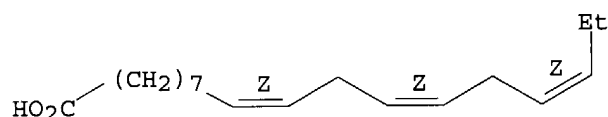
(metabolism of, by lungs in anaphylaxis)  
 IT 57-10-3, Palmitic acid 57-11-4, Stearic acid 60-33-3,  
**Linoleic acid 463-40-1, Linolenic**  
 acid  
 (in lung in anaphylaxis)  
 IT 57-88-5, Cholesterol  
 (in lungs, in anaphylaxis)  
 IT 60-33-3, **Linoleic acid 463-40-1,**  
**Linolenic acid**  
 (in lung in anaphylaxis)  
 RN 60-33-3 HCAPLUS  
 CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 463-40-1 HCAPLUS  
 CN 9,12,15-Octadecatrienoic acid, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



=> => fil medline  
 FILE 'MEDLINE' ENTERED AT 10:52:08 ON 31 AUG 2004

FILE LAST UPDATED: 28 AUG 2004 (20040828/UP). FILE COVERS 1951 TO DATE.

On February 29, 2004, the 2004 MeSH terms were loaded. See HELP RLOAD for details. OLDMEDLINE now back to 1951.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2004 vocabulary. See <http://www.nlm.nih.gov/mesh/> and [http://www.nlm.nih.gov/pubs/techbull/nd03/nd03\\_mesh.html](http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html) for a description of changes.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all tot

L109 ANSWER 1 OF 30 MEDLINE on STN  
 AN 2003420638 MEDLINE  
 DN PubMed ID: 12897994  
 TI **Omega-3 vs. omega-6 lipid**  
 emulsions exert differential influence on neutrophils in **septic shock** patients: impact on plasma **fatty acids** and **lipid** mediator generation.  
 AU Mayer Konstantin; Fegbeutel Christine; Hattar Katja; Sibelius Ulf; Kramer Hans-Joachim; Heuer Kai-Uwe; Temmesfeld-Wollbruck Bettina; Gokorsch Stephanie; Grimminger Friedrich; Seeger Werner  
 CS Department of Internal Medicine, Justus Liebig University, Klinikstrasse 36, Giessen, Germany.. Konstantin.Mayer@innere.med.uni-giessen.de

SO Intensive care medicine, (2003 Sep) 29 (9) 1472-81.  
Journal code: 7704851. ISSN: 0342-4642.

CY United States  
DT (CLINICAL TRIAL)  
Journal; Article; (JOURNAL ARTICLE)  
(RANDOMIZED CONTROLLED TRIAL)

LA English  
FS Priority Journals  
EM 200403  
ED Entered STN: 20030909  
Last Updated on STN: 20040302  
Entered Medline: 20040301

AB OBJECTIVE: To compare the effects of a conventional **omega-6 lipid** infusion and a **fish oil** based (**omega-3**) **lipid** infusion for parenteral nutrition on neutrophil function, **lipid** mediators, and plasma free **fatty acids**. DESIGN AND SETTING: Open-label, randomized, pilot study in a university hospital medical intensive care unit and experimental laboratory. PATIENTS AND PARTICIPANTS: Ten patients with **septic shock** and eight healthy controls. INTERVENTIONS: Patients (five per group) requiring parenteral nutrition received intravenously either a **omega-3** or a **omega-6 lipid** emulsion for a 10-day period. MEASUREMENTS AND RESULTS: At baseline levels of plasma free **fatty acids** were elevated several-fold, including high concentrations of the **omega-6 lipid** precursor **arachidonic acid** (AA). Neutrophils isolated from **septic** patients displayed markedly reduced responsiveness to ex vivo stimulation, including **lipid** mediator generation [leukotrienes (LT), PAF], respiratory burst, and phosphoinositide hydrolysis signaling. Under the **omega-6 lipid** infusion regimen abnormalities in plasma free **fatty acids** and impairment of neutrophil functions persisted or worsened. In contrast, a rapid switch in the plasma free **fatty acid** fraction to predominance of the **omega-3** acids **eicosapentaenoic acid** and **docosahexaenoic acid** over AA occurred in response to **omega-3 lipid** infusion. LTB(5), in addition to LTB(4), appeared upon neutrophil stimulation originating from these patients, and neutrophil function was significantly improved in the **omega-3 lipid** group. CONCLUSIONS: **omega-3** vs. **omega-6 lipid** emulsions differentially influence the plasma free **fatty acid** profile with impact on neutrophil functions. **Lipid**-based parenteral nutrition in **septic** patients may thus exert profound influence on sequelae and status of immunocompetence and inflammation.

CT Check Tags: Comparative Study; Female; Human; Male; Support, Non-U.S. Gov't  
Adult  
Aged  
\*Fat Emulsions, Intravenous: AD, administration & dosage  
\*Fatty Acids, Nonesterified: BL, blood  
\*Fatty Acids, Omega-3: AD, administration & dosage  
\*Fatty Acids, Omega-6: AD, administration & dosage  
Fish Oils: AD, administration & dosage  
Leukotrienes: ME, metabolism  
Middle Aged  
\*Neutrophils: ME, metabolism  
Phosphatidylinositols: ME, metabolism  
Pilot Projects  
Platelet Activating Factor: ME, metabolism  
Shock, Septic: ME, metabolism

**\*Shock, Septic: TH, therapy**

Superoxides: ME, metabolism

Thromboxanes: ME, metabolism

RN 11062-77-4 (Superoxides)

CN 0 (Fat Emulsions, Intravenous); 0 (**Fatty Acids**,Nonesterified); 0 (**Fatty Acids**, Omega-3); 0 (**Fatty Acids**, Omega-6); 0 (**Fish Oils**); 0 (Leukotrienes); 0

(Phosphatidylinositols); 0 (Platelet Activating Factor); 0 (Thromboxanes)

L109 ANSWER 2 OF 30 MEDLINE on STN

AN 2003216984 MEDLINE

DN PubMed ID: 12615625

TI Parenteral nutrition with **fish oil** modulates cytokine response in patients with **sepsis**.

AU Mayer Konstantin; Gokorsch Stephanie; Fegbeutel Christine; Hattar Katja; Rosseau Simone; Walmrath Dieter; Seeger Werner; Grimminger Friedrich

CS Medizinische Klinik II, Justus-Liebig-University, Giessen, Germany..

Konstantin.Mayer@innere.med.uni-giessen.de

SO American journal of respiratory and critical care medicine, (2003 May 15) 167 (10) 1321-8.

Journal code: 9421642. ISSN: 1073-449X.

CY United States

DT (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

(RANDOMIZED CONTROLLED TRIAL)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 200306

ED Entered STN: 20030513

Last Updated on STN: 20030612

Entered Medline: 20030611

AB Infusion of **fish oil**-based (n-3)**lipids** may influence leukocyte function and plasma **lipids** in critical care patients. Twenty-one patients with **sepsis** requiring parenteral nutrition were randomized to receive an n-3 **lipid** emulsion rich in **eicosapentaenoic****acid** and **docosahexaenoic acid** or aconventional (n-6) **lipid** emulsion (index**fatty acid**: **arachidonic acid**) for 5days. The impact on plasma-free **fatty acids**,mononuclear leukocyte cytokine generation, and membrane **fatty****acid** composition was examined. Cytokine synthesis by isolated

mononuclear leukocyte was elicited by endotoxin. Before the onset of

**lipid** infusion therapy, plasma-free **fatty acid**concentrations were greatly increased in **septic** patients, with**arachidonic acid** by far surpassing**eicosapentaenoic acid** and **docosahexaenoic****acid**, a feature maintained during conventional **lipid**infusion. Within 2 days of **fish oil** infusion, freen-3 **fatty acids** increased, and the

n-3/n-6 ratio was reversed, with

rapid incorporation of n-3 **fatty****acids** into mononuclear leukocyte membranes. Generation of

proinflammatory cytokines by mononuclear leukocytes was markedly amplified

during n-6 and was suppressed during n-

3 **lipid** application. After termination of **lipid**administration, free n-3 **fatty acid**

concentrations and mononuclear leukocyte cytokine synthesis returned to

preinfusion values. Use of **lipid** infusions might allow us to

combine intravenous alimentation with differential impact on inflammatory

events and immunologic functions in patients with **sepsis**.

CT Check Tags: Comparative Study; Female; Human; Male; Support, Non-U.S.

Gov't

Critical Illness

Cytokines: AN, analysis

\*Cytokines: BI, biosynthesis

Docosahexaenoic Acids: AD, administration &amp; dosage

Eicosapentaenoic Acid: AD, administration &amp; dosage

Fat Emulsions, Intravenous: AD, administration &amp; dosage

\*Fish Oils: AD, administration &amp; dosage

Follow-Up Studies

Leukocytes, Mononuclear: ME, metabolism

\*Parenteral Nutrition: MT, methods

Sensitivity and Specificity

Sepsis: DI, diagnosis

\*Sepsis: TH, therapy

Shock, Septic: DI, diagnosis

\*Shock, Septic: TH, therapy

Treatment Outcome

RN 1553-41-9 (Eicosapentaenoic Acid); 25167-62-8  
(Docosahexaenoic Acids)CN 0 (Cytokines); 0 (Fat Emulsions, Intravenous); 0 (Fish  
Oils)

L109 ANSWER 3 OF 30 MEDLINE on STN

AN 2002492402 MEDLINE

DN PubMed ID: 12353921

TI A **fish oil** emulsion used for parenteral nutrition  
attenuates monocyte-endothelial interactions under flow.AU Nohe Boris; Ruoff Heinrich; Johannes Tanja; Zanke Christof; Unertl Klaus;  
Dieterich Hans-JuergenCS Department of Anesthesiology and Critical Care, University Hospital  
Tuebingen, Germany.SO Shock (Augusta, Ga.), (2002 Sep) 18 (3) 217-22.  
Journal code: 9421564. ISSN: 1073-2322.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200312

ED Entered STN: 20021001

Last Updated on STN: 20021213

Entered Medline: 20031218

AB Monocyte adhesion contributes to perfusion abnormalities, tissue damage, and activation of the coagulation system seen during trauma, **shock**, or overwhelming inflammation. This study was performed to determine whether an intravenous **fish oil** emulsion used for parenteral nutrition attenuates monocyte-endothelial interactions under flow and reduces procoagulant activity, measured as tissue factor (TF) expression on adherent monocytes in vitro. Endothelial cell monolayers were incubated with either an intravenous **fish oil** emulsion or a conventional **omega-6 lipid** emulsion at 0.05 to 1 mg/ml for 24 h. Six hours following activation with TNFalpha (25 ng/ml), expression of endothelial cell adhesion molecules was measured by flow cytometry. Adhesion of isolated monocytes to pretreated endothelium was examined in a parallel plate flow chamber at a shear stress of 1.5 dynes/cm<sup>2</sup>. Following perfusion, the cells were cocultured for an additional 4 h and TF expression on monocytes was determined by flow cytometry. In contrast to **omega-6 lipids**, **fish oil** down-regulated E-selectin, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1 in a dose-dependent manner. P-selectin, however, remained unchanged. In addition, firm adhesion was reduced to 54%, whereas rolling interactions remained unchanged. **Fish oil** exhibited no effect on the TF expression on cocultured monocytes. We conclude that intravenous

**fish oil** emulsions reduce both endothelial cell adhesion molecule expression and monocyte adhesion. However, under postcapillary flow conditions, rolling interactions via P-selectin remain unaltered. The functional importance of this effect is illustrated by the corresponding upregulation of TF in response to residual monocyte-endothelial interactions.

CT Check Tags: Human; Support, Non-U.S. Gov't

Cell Adhesion: DE, drug effects

Cell Adhesion Molecules: ME, metabolism

Cells, Cultured

Coculture

Cytokines: PD, pharmacology

\*Emulsions: PD, pharmacology

\*Endothelium: CY, cytology

\*Endothelium: DE, drug effects

Endothelium: ME, metabolism

**\*Fish Oils: PD, pharmacology**

\*Monocytes: CY, cytology

\*Monocytes: DE, drug effects

Monocytes: ME, metabolism

\*Parenteral Nutrition

Thromboplastin: ME, metabolism

RN 9035-58-9 (Thromboplastin)

CN 0 (Cell Adhesion Molecules); 0 (Cytokines); 0 (Emulsions); 0 (**Fish Oils**)

L109 ANSWER 4 OF 30 MEDLINE on STN

AN 2002162753 MEDLINE

DN PubMed ID: 11895156

TI Parenteral nutrition with n-3 **lipids** in **sepsis**.

AU Mayer K; Grimm H; Grimmering F; Seeger W

CS Medizinische Klinik II der Justus-Liebig-Universitat Giessen, Germany..  
Konstantin.mayer@innere.med.unigiessen.de

SO British journal of nutrition, (2002 Jan) 87 Suppl 1 S69-75. Ref: 67  
Journal code: 0372547. ISSN: 0007-1145.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200204

ED Entered STN: 20020317

Last Updated on STN: 20020419

Entered Medline: 20020418

AB Dietary supplements of n-3 **fatty**

**acids** have long been used to influence chronic inflammatory disorders. Recent studies with an immune-enhancing diet partly based on **n-3 fatty acids** report beneficial

effects in patients with acute hyper-inflammatory diseases, such as the **sepsis** syndrome or adult respiratory distress syndrome (ARDS).

The possible suppression of exaggerated leucocyte activity, the improvement of microcirculatory events, as well as the opportunity to administer intravenous **lipids** enriched in **n-3**

**fatty acids** signal the possibility of a combination of parenteral caloric support and pharmacological intervention. Using parenteral administration of **fish oil**-based

**lipids**, a new rapid and highly effective anti-inflammatory agent may allow the option to alter the immune status in hyper-inflammatory diseases such as **sepsis** and ARDS.

CT Check Tags: Human

**\*Fatty Acids, Omega-3: TU, therapeutic use**

Inflammation: TH, therapy

\*Parenteral Nutrition: MT, methods  
Respiratory Distress Syndrome, Adult: TH, therapy  
**Sepsis Syndrome: PP, physiopathology**  
\*Sepsis Syndrome: TH, therapy

CN 0 (**Fatty Acids, Omega-3**)

L109 ANSWER 5 OF 30 MEDLINE on STN

AN 2001634858 MEDLINE

DN PubMed ID: 11240338

TI Parenteral supplementation with a **fish-oil** emulsion  
prolongs survival and improves rat lymphocyte function during  
**sepsis**.

CM Comment in: Nutrition. 2001 Feb;17(2):158-60. PubMed ID: 11240346

AU Lanza-Jacoby S; Flynn J T; Miller S

CS Department of Surgery, Thomas Jefferson University, Jefferson Medical  
College, Philadelphia, Pennsylvania 19107, USA.. susan.lanza-  
jacoby@mail.tju.edu

SO Nutrition (Burbank, Los Angeles County, Calif.), (2001 Feb) 17 (2) 112-6.  
Journal code: 8802712. ISSN: 0899-9007.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200111

ED Entered STN: 20011105

Last Updated on STN: 20011105

Entered Medline: 20011101

AB Nutritional intervention with **omega-3 fatty**

**acids** during trauma and infection has been shown to improve the  
clinical outcome of patients and the survival rate in laboratory animals.  
This study evaluated the effects of parenteral administration of  
**lipid** emulsions containing **fish oil** (FO) or  
soybean oil (SBO) on survival and T-lymphocyte response during  
**sepsis**. Male Sprague-Dawley rats (250-275 g) were prepared for  
parenteral feeding 4 d before inducing **sepsis** by cecal ligation  
and puncture (CLP). Standard resuscitation was provided with normal  
saline. Thirty minutes after completing CLP, sham control or CLP rats  
were infused continuously with saline or a parenteral diet containing SBO  
or a 1:1 FO:SBO emulsion. The survival rate was significantly improved in  
rats receiving the FO-supplemented diet, with 50% alive by 120 h in  
comparison with the saline-infused, chow-fed rats (0% alive by 120 h) or  
the SBO-fed rats (12% alive at 120 h). The T-lymphocyte response was  
evaluated at 24 h after CLP. **Sepsis** led to a decline in  
lymphocyte proliferation in rats infused with saline or the SBO emulsion,  
which was associated with a greater release of splenocyte interleukin-10,  
transforming growth factor-beta and prostaglandin E2. Administering the  
1:1 FO:SBO parenteral diet during **sepsis** improved the survival  
rate and prevented the **sepsis**-induced suppression of lymphocyte  
proliferation and interleukin-2 release. The FO effect on lymphocyte  
function was associated with decreased splenocyte release of transforming  
growth factor-beta and prostaglandin E2.

CT Check Tags: Male

Animals

Cecum: SU, surgery

Dietary Supplements

Dinoprostone: BI, biosynthesis

\*Fat Emulsions, Intravenous: AD, administration & dosage

Fat Emulsions, Intravenous: CH, chemistry

\***Fish Oils: AD, administration & dosage**

**Fish Oils: PD, pharmacology**

Interleukin-10: SE, secretion

Interleukin-2: SE, secretion

Ligation



Lymphocyte Activation

\*Parenteral Nutrition

Punctures

Rats

Rats, Sprague-Dawley

**Sepsis: IM, immunology**

**Sepsis: MO, mortality**

**\*Sepsis: TH, therapy**

Soybean Oil: AD, administration & dosage

Soybean Oil: PD, pharmacology

Survival Analysis

\*T-Lymphocytes: IM, immunology

Transforming Growth Factor beta: BL, blood

Transforming Growth Factor beta: SE, secretion

RN 130068-27-8 (Interleukin-10); 363-24-6 (Dinoprostone); 8001-22-7 (Soybean Oil)

CN 0 (Fat Emulsions, Intravenous); 0 (**Fish Oils**); 0 (Interleukin-2); 0 (Transforming Growth Factor beta)

L109 ANSWER 6 OF 30 MEDLINE on STN

AN 2001608625 MEDLINE

DN PubMed ID: 11685569

TI Immunonutrition--supplementary amino acids and **fatty acids** ameliorate immune deficiency in critically ill patients.

AU Grimm H; Kraus A

CS Department of General and Thoracic Surgery, Justus Liebig University, Rudolf-Buchheim-Strasse 7, 35385 Giessen, Germany..  
helmut.grimm@chiru.med.uni-giessen.de

SO Langenbeck's archives of surgery / Deutsche Gesellschaft fur Chirurgie, (2001 Aug) 386 (5) 369-76. Ref: 70  
Journal code: 9808285. ISSN: 1435-2443.

CY Germany: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200112

ED Entered STN: 20011102

Last Updated on STN: 20020123

Entered Medline: 20011205

AB BACKGROUND: Immunonutrition with **omega-3 fatty**

**acids** and the "conditionally essential" amino acids arginine, glutamine, cysteine, and taurine can enhance the immune response in critically ill patients. This is due to the immunomodulating properties of these nutrients. Immunonutrition is especially important when a patient's immune response is compromised, as is the case post-operatively or after trauma. Immune deficiency is severely aggravated in **sepsis** and the systemic inflammatory response syndrome (SIRS).

The resulting metabolic stress is characterized by glycolysis, lipolysis, and proteolysis, which may escalate to an hypercatabolic response or "autocannibalism." Catabolic metabolism results in insufficiency of both specific and unspecific immunocompetent cells. CONCLUSIONS:

Immunonutrition should be started early in such patients for an optimal beneficial effect, preferably via the enteral route. It should include

**medium chain** and long chain **triglycerides**, polyunsaturated **omega-3** and **omega-6**

**fatty acids** (in the ratio 1:2), olive oil, and

conventional amino acid preparations supplemented with the conditionally essential amino acids arginine, glutamine, cysteine, and taurine.

CT Check Tags: Human

\*Adjuvants, Immunologic: TU, therapeutic use

\*Amino Acids, Essential: TU, therapeutic use

Date  
no good

\*Critical Illness  
 \*Dietary Supplements  
 Endotoxins: BL, blood  
 Enteral Nutrition: MT, methods  
 \*Fatty Acids, Omega-3: TU, therapeutic use  
 \*Immunocompromised Host: PH, physiology  
 \*Immunologic Deficiency Syndromes: DH, diet therapy  
 Immunologic Deficiency Syndromes: ET, etiology  
 Immunologic Deficiency Syndromes: ME, metabolism  
 Lipids: ME, metabolism  
 Proteins: DE, drug effects  
 Proteins: ME, metabolism  
 Signal Transduction: DE, drug effects

CN 0 (Adjuvants, Immunologic); 0 (Amino Acids, Essential); 0 (Endotoxins); 0 (Fatty Acids, Omega-3); 0 (Lipids); 0 (Proteins)

L109 ANSWER 7 OF 30 MEDLINE on STN

AN 2001140272 MEDLINE

DN PubMed ID: 11133172

TI Beneficial effect(s) of **n-3 fatty**

**acids** in cardiovascular diseases: but, why and how?.

CM Erratum in: Prostaglandins Leukot Essent Fatty Acids 2001 Jan;64(1):74

AU Das U N

CS EFA Sciences LLC, 1420 Providence Highway, Norwood, MA 02062, USA..  
 undurti@hotmail.com

SO Prostaglandins, leukotrienes, and essential fatty acids, (2000 Dec) 63 (6)  
 351-62. Ref: 143

Journal code: 8802730. ISSN: 0952-3278.

CY Scotland: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, ACADEMIC)

LA English

FS Priority Journals

EM 200103

ED Entered STN: 20010404

Last Updated on STN: 20011018

Entered Medline: 20010308

AB Low rates of coronary heart disease was found in Greenland Eskimos and Japanese who are exposed to a diet rich in **fish oil**.

Suggested mechanisms for this cardio-protective effect focused on the effects of **n-3 fatty acids** on

eicosanoid metabolism, inflammation, beta oxidation, endothelial dysfunction, cytokine growth factors, and gene expression of adhesion molecules; But, none of these mechanisms could adequately explain the beneficial actions of **n-3 fatty**

**acids**. One attractive suggestion is a direct cardiac effect of **n-3 fatty acids** on arrhythmogenesis.

**N-3 fatty acids** can modify Na+

channels by directly binding to the channel proteins and thus, prevent ischemia-induced ventricular fibrillation and sudden cardiac death.

Though this is an attractive explanation, there could be other actions as well. **N-3 fatty acids** can inhibit

the synthesis and release of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNFalpha) and interleukin-1 (IL-1) and IL-2 that are released during the early course of ischemic heart disease. These cytokines decrease myocardial contractility and induce myocardial damage, enhance the production of free radicals, which can also suppress myocardial function. Further, **n-3 fatty**

**acids** can increase parasympathetic tone leading to an increase in heart rate variability and thus, protect the myocardium against ventricular arrhythmias. Increased parasympathetic tone and

acetylcholine, the principle vagal neurotransmitter, significantly attenuate the release of TNF, IL-1beta, IL-6 and IL-18. Exercise enhances parasympathetic tone, and the production of anti-inflammatory cytokine IL-10 which may explain the beneficial action of exercise in the prevention of cardiovascular diseases and diabetes mellitus. TNFalpha has neurotoxic actions, where as **n-3 fatty acids** are potent neuroprotectors and brain is rich in these **fatty acids**. Based on this, it is suggested that the principle mechanism of cardioprotective and neuroprotective action(s) of **n-3 fatty acids** can be due to the suppression of TNFalpha and IL synthesis and release, modulation of hypothalamic-pituitary-adrenal anti-inflammatory responses, and an increase in acetylcholine release, the vagal neurotransmitter. Thus, there appears to be a close interaction between the central nervous system, endocrine organs, cytokines, exercise, and dietary **n-3 fatty acids**. This may explain why these **fatty acids** could be of benefit in the management of conditions such as **septicemia** and **septic shock**, Alzheimer's disease, Parkinson's disease, inflammatory bowel diseases, diabetes mellitus, essential hypertension and atherosclerosis.

CT

Check Tags: Human

Acetylcholine: PH, physiology

Animals

Arrhythmia: EP, epidemiology

Arrhythmia: PC, prevention &amp; control

Brain: PP, physiopathology

Cardiovascular Diseases: DH, diet therapy

Cardiovascular Diseases: EP, epidemiology

\*Cardiovascular Diseases: PC, prevention &amp; control

Cell Adhesion Molecules: BI, biosynthesis

Cell Adhesion Molecules: GE, genetics

Cell Division: DE, drug effects

Clinical Trials

Cohort Studies

Cytokines: ME, metabolism

Dietary Fats: AD, administration &amp; dosage

\*Dietary Fats: PD, pharmacology

Dietary Fats: TU, therapeutic use

Eicosanoids: ME, metabolism

Endothelium, Vascular: DE, drug effects

Endothelium, Vascular: ME, metabolism

Exercise

Fatty Acids, Omega-3: AD, administration &amp; dosage

\*Fatty Acids, Omega-3: PD, pharmacology

Fatty Acids, Omega-3: TU, therapeutic use

Fatty Acids, Unsaturated: ME, metabolism

Fish Oils: AD, administration &amp; dosage

\*Fish Oils: PD, pharmacology

Fish Oils: TU, therapeutic use

Gene Expression Regulation: DE, drug effects

Greenland: EP, epidemiology

Heart: DE, drug effects

Hemostasis: DE, drug effects

Hypothalamo-Hypophyseal System: DE, drug effects

Hypothalamo-Hypophyseal System: PP, physiopathology

Inflammation: DT, drug therapy

Inflammation: ME, metabolism

Inflammation: PC, prevention &amp; control

Inuits

Japan: EP, epidemiology

Lipids: ME, metabolism

Models, Biological

Myocardium: ME, metabolism

Oxidation-Reduction  
 Oxidative Stress  
 Parasympathetic Nervous System: DE, drug effects  
 Pituitary-Adrenal System: DE, drug effects  
 Pituitary-Adrenal System: PP, physiopathology  
 Rats  
 Sodium Channels: DE, drug effects  
 Vagus Nerve: PP, physiopathology

RN 51-84-3 (Acetylcholine)  
 CN 0 (Cell Adhesion Molecules); 0 (Cytokines); 0 (Dietary Fats); 0  
 (Eicosanoids); 0 (**Fatty Acids, Omega-**  
**3**); 0 (**Fatty Acids, Unsaturated**); 0 (**Fish Oils**); 0 (**Lipids**); 0 (Sodium Channels)

L109 ANSWER 8 OF 30 MEDLINE on STN

AN 2001028371 MEDLINE

DN PubMed ID: 10850941

TI **Fish oil** modulates macrophage P44/P42  
 mitogen-activated protein kinase activity induced by lipopolysaccharide.

AU Lo C J; Chiu K C; Fu M; Chu A; Helton S

CS Department of Surgery, University of California, Los Angeles, USA..  
 clo@mednet.ucla.edu

SO JPEN. Journal of parenteral and enteral nutrition, (2000 May-Jun) 24 (3)  
 159-63.

Journal code: 7804134. ISSN: 0148-6071.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200011

ED Entered STN: 20010322

Last Updated on STN: 20010322

Entered Medline: 20001121

AB BACKGROUND: Mitogen-activated protein kinase (MAPK) cascades represent a major signal system to transduce extracellular signals into cellular responses. Overactivity of MAPK has been implicated in the development of many diseases, including cancer and **sepsis**. This study investigated the hypothesis that **fish oil** altered the membrane phospholipid composition and modulated MAPK activity. METHODS: RAW 264.7 cells, a mouse macrophage (Mphi) cell line, were grown in **eicosapentaenoic acid** (EPA)-rich media (114 micromol/L) for 48 hours. Mphi were washed and exposed to Escherichia coli lipopolysaccharide (LPS; 1 microg/mL) for 10 minutes. Both total and activated (phosphorylated) portions of MAPK (P44 and P42) were determined by Western blot assays. AP-1 transcription factor activity was determined by electrophoretic mobility gel shift assays (EMSA). Mphi tumor necrosis factor (TNF) mRNA expression was measured by Northern blot assays. RESULTS: LPS stimulation induced RAW cell phosphorylation of P44/P42. In contrast, RAW cells grown in EPA-rich media had less P44/P42 activation in the presence of LPS. Total P44/P42 were not affected by EPA or LPS. Similarly, EPA also inhibited AP-1 activity. Inhibition of P44/P42 activity with PD98059 reduced both AP-1 activity and TNF mRNA expression of LPS-stimulated Mphi. CONCLUSIONS: Our data suggest that **fish oil** regulates macrophage proinflammatory gene activation, at least in part, by modulating the MAPK activity.

CT Check Tags: Support, Non-U.S. Gov't  
 Animals

**Arachidonic Acids**

Blotting, Western

Cell Line

Down-Regulation

Electrophoresis

Escherichia coli

**\*Fish Oils: PD, pharmacology**  
 Lipopolysaccharides: PD, pharmacology  
 Macrophages: DE, drug effects  
 Macrophages: EN, enzymology  
 \*Macrophages: PH, physiology  
**Membrane Lipids: CH, chemistry**  
 Mice  
 Mitogen-Activated Protein Kinases: DE, drug effects  
 \*Mitogen-Activated Protein Kinases: ME, metabolism  
 RNA, Messenger  
 Tumor Necrosis Factor: BI, biosynthesis  
 Tumor Necrosis Factor: GE, genetics

CN 0 (**Arachidonic Acids**); 0 (**Fish Oils**);  
 0 (Lipopolysaccharides); 0 (Membrane **Lipids**); 0 (RNA,  
 Messenger); 0 (Tumor Necrosis Factor); EC 2.7.1.37 (Mitogen-Activated  
 Protein Kinases)

L109 ANSWER 9 OF 30 MEDLINE on STN

AN 2000223123 MEDLINE

DN PubMed ID: 10758365

TI Effects of parenteral infusion with **fish-oil** or  
 safflower-oil emulsion on hepatic **lipids**, plasma amino acids,  
 and inflammatory mediators in **septic** rats.

CM Comment in: Nutrition. 2000 Apr;16(4):308-9. PubMed ID: 10758371

AU Chao C Y; Yeh S L; Lin M T; Chen W J

CS Institute of Nutrition and Health Science, Taipei Medical College, Taipei,  
 Taiwan, People's Republic of China.

SO Nutrition (Burbank, Los Angeles County, Calif.), (2000 Apr) 16 (4) 284-8.  
 Journal code: 8802712. ISSN: 0899-9007.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200007

ED Entered STN: 20000720

Last Updated on STN: 20000720

Entered Medline: 20000710

AB This study was designed to investigate the effects of preinfusion with  
 total parenteral nutrition (TPN) using **fish-oil** (FO)  
 versus safflower-oil (SO) emulsion as fat sources on hepatic  
**lipids**, plasma amino-acid profiles, and inflammatory-related  
 mediators in **septic** rats. Normal rats, with internal jugular  
 catheters, were assigned to two different groups and received TPN. TPN  
 provided 300 kcal. kg<sup>-1</sup>. d<sup>-1</sup>, with 40% of the non-protein energy as  
 fat. All TPN solutions were isonitrogenous and identical in nutrient  
 composition except for the fat emulsion, which was made of SO or FO.  
 After receiving TPN for 6 d, each group of rats was further divided into  
 control and **sepsis** subgroups. **Sepsis** was induced by  
 cecal ligation and puncture; control rats received sham operation. All  
 rats were classified into four groups as follows: FO control group (FOC; n  
 = 7), FO **sepsis** group (FOS; n = 8), SO control group (SOC; n =  
 8), and SO **sepsis** group (SOS; n = 9). The results of the study  
 demonstrated that plasma concentrations of triacylglycerol and  
 non-esterified **fatty acids** did not differ between the  
 FO and SO groups, regardless of whether the animals were **septic**.  
 SOS had significantly higher total **lipids** and cholesterol  
 content in the liver than did the SOC group. The FOS group, however,  
 showed no difference from the FOC group. Plasma leucine and isoleucine  
 levels were significantly lower in the SOS group than in the SOC group,  
 whereas no difference in these two amino acids was observed between the  
 FOC and FOS groups. Plasma arginine levels were significantly lower in  
 both **septic** groups than in the groups without **sepsis**  
 when either FO or SO was infused. Plasma glutamine levels, however, did

not differ across groups. No differences in interleukin-1beta, interleukin-6, tumor necrosis factor-alpha, or leukotriene B(4) concentrations in peritoneal lavage fluid were observed between the two **septic** groups. These results suggest that catabolic reaction in **septic** rats preinfused with FO is not as obvious as those preinfused with SO. Compared with SO emulsion, TPN with FO emulsion prevents liver fat accumulation associated with **sepsis**. However, parenterally administered FO had no beneficial effect in lowering cytokines and LTB(4) levels in peritoneal lavage fluid in **septic** rats induced by cecal ligation and puncture.

CT Check Tags: Male; Support, U.S. Gov't, Non-P.H.S.

\*Amino Acids: BL, blood

Animals

\*Cytokines: BL, blood

Disease Models, Animal

Fat Emulsions, Intravenous: AD, administration & dosage

\*Fish Oils: AD, administration & dosage

Lipids: BL, blood

\*Lipids: ME, metabolism

\*Liver: ME, metabolism

\*Parenteral Nutrition, Total

Rats

Rats, Wistar

\*Safflower Oil: AD, administration & dosage

Sepsis: BL, blood

Sepsis: TH, therapy

RN 8001-23-8 (Safflower Oil)

CN 0 (Amino Acids); 0 (Cytokines); 0 (Fat Emulsions, Intravenous); 0 (

Fish Oils); 0 (Lipids)

L109 ANSWER 10 OF 30 MEDLINE on STN

AN 2000207435 MEDLINE

DN PubMed ID: 10743032

TI [Immunonutrition with **omega-3-fatty acids**. Are new anti-inflammatory strategies in sight?].  
Immunonutrition mit **Omega-3** Fettsauren. Sind neue  
anti-inflammatorische Strategien in Sicht?.

AU Heller A; Koch T

CS Klinik und Poliklinik fur Anesthesiologie und Intensivtherapie,  
Universitätsklinikum Carl Gustav Carus, Technischen Universität Dresden..  
heller-a@rcs.urz.tu-dresden.de

SO Zentralblatt fur Chirurgie, (2000) 125 (2) 123-36. Ref: 126  
Journal code: 0413645. ISSN: 0044-409X.

CY GERMANY: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA German

FS Priority Journals

EM 200005

ED Entered STN: 20000512

Last Updated on STN: 20000512

Entered Medline: 20000504

AB In the early phase of **sepsis** and SIRS an overwhelming activation of humoral and cellular mediator systems can alter vascular resistance and causes capillary leakage increasing the risk of organ dysfunction. **omega-6-arachidonic acid** is released from **lipid** pools of cellular membranes during inflammation and is metabolized to pro-inflammatory prostaglandins and leukotriens, which are key mediators in the pathogenesis of organ dysfunction. **omega-3-eicosapentaenoic acid**-derived **lipid** mediators present altered biologic effects. Thus, **omega-3-fatty acid** application

enables anti-inflammatory intervention on the level of **lipid** mediators. The current article reviews experimental and clinical data on **omega-3-fatty acids**. Besides the decrease of pro-inflammatory mediators, **fish oil** supplementation lowered post operative infection rates and showed a tendency to reduce hospital stay in surgical patients. It is believed that the decreased formation of LTB4 and TXA2 during **sepsis** after administration of **omega-3-fatty acids** accounts for improved microcirculatory perfusion and declined lactate acidosis.

CT Check Tags: Human

Animals

Capillary Leak Syndrome: IM, immunology

Capillary Leak Syndrome: TH, therapy

English Abstract

**\*Fatty Acids, Omega-3: AD, administration & dosage**

Inflammation Mediators: BL, blood

Intensive Care

**Sepsis Syndrome: IM, immunology**

**\*Sepsis Syndrome: TH, therapy**

Treatment Outcome

CN 0 (**Fatty Acids, Omega-3**); 0  
(Inflammation Mediators)

L109 ANSWER 11 OF 30 MEDLINE on STN

AN 1998222123 MEDLINE

DN PubMed ID: 9561339

TI **Lipid** mediators in inflammatory disorders.

AU Heller A; Koch T; Schmeck J; van Ackern K

CS Department of Anaesthesiology and Intensive Care Medicine, University of Dresden, Germany.. heller@rumms.uni-mannheim.de

SO Drugs, (1998 Apr) 55 (4) 487-96. Ref: 46

Journal code: 7600076. ISSN: 0012-6667.

CY New Zealand

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199806

ED Entered STN: 19980625

Last Updated on STN: 19980625

Entered Medline: 19980618

AB During the past few decades, intensive collaborative research in the fields of chronic and acute inflammatory disorders has resulted in a better understanding of the pathophysiology and diagnosis of these diseases. Modern therapeutic approaches are still not satisfactory and **shock, sepsis** and multiple organ failure remain the great challenge in intensive care medicine. However, the treatment of inflammatory diseases like rheumatoid arthritis, ulcerative colitis or psoriasis also represents an unresolved problem. Many factors contribute to the complex course of inflammatory reactions. Microbiological, immunological and toxic agents can initiate the inflammatory response by activating a variety of humoral and cellular mediators. In the early phase of inflammation, excessive amounts of interleukins and **lipid** -mediators are released and play a crucial role in the pathogenesis of organ dysfunction. **Arachidonic acid** (AA), the mother substance of the pro-inflammatory eicosanoids, is released from membrane phospholipids in the course of inflammatory activation and is metabolised to prostaglandins and leukotrienes. Various strategies have been evaluated to control the excessive production of **lipid** mediators on different levels of biochemical pathways, such as inhibition of phospholipase A2, the trigger enzyme for release of AA, blockade of

cyclooxygenase and lipoxygenase pathways and the development of receptor antagonists against platelet activating factor and leukotrienes. Some of these agents exert protective effects in different inflammatory disorders such as **septic** organ failure, rheumatoid arthritis or asthma, whereas others fail to do so. Encouraging results have been obtained by dietary supplementation with long chain **omega-3 fatty acids** like **eicosapentaenoic acid** (EPA). In states of inflammation, EPA is released to compete with AA for enzymatic metabolism inducing the production of less inflammatory and chemotactic derivatives.

CT Check Tags: Human

Animals

**\*Arachidonic Acid: ME, metabolism**

Cyclooxygenase Inhibitors: TU, therapeutic use

**Fatty Acids, Omega-3: TU, therapeutic use**

\*Inflammation: ME, metabolism

Inflammation: TH, therapy

\*Inflammation Mediators: ME, metabolism

Leukotriene Antagonists

**\*Lipids: ME, metabolism**

Lipoxygenase Inhibitors: TU, therapeutic use

Phospholipases A: AI, antagonists & inhibitors

Phospholipids: ME, metabolism

Platelet Activating Factor: AI, antagonists & inhibitors

Prostaglandins: ME, metabolism

RN 506-32-1 (Arachidonic Acid)

CN 0 (Cyclooxygenase Inhibitors); 0 (Fatty Acids, Omega-3); 0 (Inflammation Mediators); 0 (Leukotriene Antagonists); 0 (Lipids); 0 (Lipoxygenase Inhibitors); 0 (Phospholipids); 0 (Platelet Activating Factor); 0 (Prostaglandins); EC 3.1.1.- (Phospholipases A)

L109 ANSWER 12 OF 30 MEDLINE on STN

AN 1998219122 MEDLINE

DN PubMed ID: 9558431

TI [Pharmacologic aspects of polyunsaturated **fatty acids** in parenteral nutrition].

Pharmakologische Aspekte von mehrfach ungesättigten Fettsäuren in der parenteralen Ernährung.

AU Heller A; Koch T

CS Institut für Anesthesiologie und Operative Intensivmedizin, Fakultät für Klinische Medizin Mannheim, Universität Heidelberg.. heller@rumms.uni-mannheim.de

SO Anesthesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie : AINS, (1998 Feb) 33 (2) 77-87. Ref: 61  
Journal code: 9109478. ISSN: 0939-2661.

CY GERMANY: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA German

FS Priority Journals

EM 199806

ED Entered STN: 19980625

Last Updated on STN: 19980625

Entered Medline: 19980616

AB Despite immense progress in intensive-care medicine, mortality rates of 30-70% in **sepsis** and SIRS are still an unresolved problem. Particularly the failure of respiratory and other vital functions is a major cause of death. Besides infectious stimuli (viruses, bacteria, fungi) a variety of non-infectious triggers (tissue damage, immune complexes, complement activation, etc.) can initiate the development of organ failure. These inflammatory reactions aim physiologically towards



inactivation and removal of the stimulating agents as well as the induction of reparative processes. In states of prolonged activation of humoral and cellular mediator systems the natural host defence mechanisms react in an uncontrolled manner causing tissue damage and organ failure. So far there are no efficient therapeutic strategies to influence these complex inflammatory reactions. In the development of SIRS and **sepsis**, pro-inflammatory **lipid** mediators play a crucial role. **Omega-3-fatty acids** (**omega-3**-PUFAs) have shown anti-inflammatory and antithrombotic properties in a great number of experimental and clinical studies. These effects seem to be related to the uptake of **eicosapentaenoic acid** (EPA) into cellular membrane **lipid** pools and its subsequent metabolism. After inflammatory activation EPA is released besides **arachidonic acid** (AA) and competes with AA for metabolism via the cyclo- and lipoxygenase pathway. Compared to AA the derivatives of EPA have less pro-inflammatory and chemotactic characteristics. With regard to prophylactic and therapeutic consequences it appears reasonable to supplement **omega-3**-PUFAs to attenuate the inflammatory response by modulating the generation of **lipid** mediators during inflammation.

CT Check Tags: Human

Animals

Eicosanoids: PD, pharmacology

English Abstract

\*Fat Emulsions, Intravenous: PD, pharmacology

\***Fatty Acids, Omega-3: PD, pharmacology**

Inflammation Mediators: BL, blood

Multiple Organ Failure: IM, immunology

\***Sepsis Syndrome: IM, immunology**

CN 0 (Eicosanoids); 0 (Fat Emulsions, Intravenous); 0 (**Fatty Acids, Omega-3**); 0 (Inflammation Mediators)

L109 ANSWER 13 OF 30 MEDLINE on STN

AN 97239214 MEDLINE

DN PubMed ID: 9084876

TI Reevaluation of the effect of a high alpha-linolenate and a high linoleate diet on antigen-induced antibody and anaphylactic responses in mice.

AU Oh-hashii K; Watanabe S; Kobayashi T; Okuyama H

CS Department of Biological Chemistry, Faculty of Pharmaceutical Sciences, Nagoya City University, Japan.

SO Biological & pharmaceutical bulletin, (1997 Mar) 20 (3) 217-23.

Journal code: 9311984. ISSN: 0918-6158.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199706

ED Entered STN: 19970630

Last Updated on STN: 19970630

Entered Medline: 19970619

AB Previously, we reported that a high alpha-linolenate [18:3(n-3)] diet compared with a high linoleate [18:2(n-6)] diet suppressed the anti-egg albumin (EA) immunoglobulin E (IgE) antibody response in mice. Because of relatively high background values obtained with the method used previously, we used an improved ELISA and once again determined serum IgE levels. In contrast to our previous results, the serum level of anti-dinitrophenyl specific (anti-DNP) as well as total IgE in mice immunized with DNP-antigen was slightly but significantly higher in the high alpha-linolenate diet group than in the high linoleate diet group. Anti-DNP IgG1 and IgG2a antibody responses were not significantly different in mice fed these diets. Indomethacin administration during immunization tended to enhance the IgE antibody responses. The mortality

of mice from antigen-induced anaphylactic **shock** was significantly lower in the high alpha-linolenate diet group than in the high linoleate diet group; however, there was no difference between the groups in terms of vascular permeability and histamine levels. Thus, the high alpha-linolenate diet enhances the IgE antibody response slightly without affecting either the IgG antibody response, vascular permeability or histamine release. The high alpha-linolenate diet possibly suppresses anaphylactic **shock** by reducing the synthesis of **lipid** mediators such as eicosanoids and platelet-activating factor.

CT Check Tags: Female  
 Anaphylaxis: MO, mortality  
 \*Anaphylaxis: PP, physiopathology  
 Animals  
 \*Antibody Formation: DE, drug effects  
 Capillary Permeability: DE, drug effects  
 Capillary Permeability: PH, physiology  
 Cyclooxygenase Inhibitors: PD, pharmacology  
 \*Diet  
 Dinitrophenols: IM, immunology  
 Enzyme-Linked Immunosorbent Assay  
**Fatty Acids: BL, blood**  
 Histamine Release: DE, drug effects  
 Immunoglobulin E: AN, analysis  
 Indomethacin: PD, pharmacology  
**\*Linoleic Acids: PD, pharmacology**  
**Lipids: BL, blood**  
 Mice  
 Mice, Inbred C3H  
**\*alpha-Linolenic Acid: PD, pharmacology**  
 RN 37341-29-0 (Immunoglobulin E); 463-40-1 (alpha-Linolenic Acid);  
 53-86-1 (Indomethacin)  
 CN 0 (Cyclooxygenase Inhibitors); 0 (Dinitrophenols); 0 (Fatty  
**Acids); 0 (Linoleic Acids); 0 (Lipids**  
 )

L109 ANSWER 14 OF 30 MEDLINE on STN  
 AN 96131218 MEDLINE  
 DN PubMed ID: 8523633  
 TI Effect of different combinations of dietary additives on bacterial translocation and survival in gut-derived **sepsis**.  
 AU Gennari R; Alexander J W; Eaves-Pyles T  
 CS University of Cincinnati Medical Center, Department of Surgery, OH 45267-0558, USA.  
 NC AI-12936 (NIAID)  
 SO JPEN. Journal of parenteral and enteral nutrition, (1995 Jul-Aug) 19 (4) 319-25.  
 Journal code: 7804134. ISSN: 0148-6071.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199601  
 ED Entered STN: 19960219  
 Last Updated on STN: 19980206  
 Entered Medline: 19960125  
 AB BACKGROUND: Dietary arginine, glutamine, and **fish oil** each have been shown to improve resistance to infection. The purpose of this study was to assess the potential benefit of different combinations and amounts of these components on bacterial translocation and related mortality during gut-derived **sepsis**. METHODS: Balb/c mice were fed for 10 days with an AIN-76A diet supplemented with different combinations and percentages of arginine, glutamine, glycine, **fish oil**, and **medium-chain triglycerides**.

Controls were fed a complete AIN-76A diet or chow. After 10 days of feeding, all animals were transfused. On day 15, the animals were gavaged with 10(10) 111In-radiolabeled or unlabeled Escherichia coli and given a 30% burn injury. Animals gavaged with unlabeled bacteria were observed for survival (n = 317). Groups that showed the best survival as well as control groups were gavaged with labeled bacteria and killed 4 hours postburn (n = 60) for harvest of mesenteric lymph nodes, liver and spleen. RESULTS: Mice fed diets enriched with 5% **fish oil** + 2% arginine, 2% arginine + 2% glutamine, or 5% **fish oil** + 2% glutamine had higher survival than control groups. The animals fed **fish oil**+glutamine had significantly reduced translocation to the liver and spleen. Animals fed arginine+glutamine had an enhanced ability to kill translocated organisms in the liver compared with other groups. **Fish oil**+arginine improved both barrier function and microbial killing. CONCLUSIONS: Feeding with arginine+glutamine, **fish oil**+arginine, or **fish oil**+glutamine supplemented diets positively affects the outcome in a gut-derived **sepsis** model.

CT Check Tags: Female; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.

Animals

Arginine: PD, pharmacology

\*Bacterial Translocation: DE, drug effects

Digestive System: MI, microbiology

Escherichia coli: DE, drug effects

\*Escherichia coli: PH, physiology

\*Escherichia coli Infections: PP, physiopathology

**Fish Oils: PD, pharmacology**

\*Food Additives: PD, pharmacology

Food, Fortified

Glutamine: PD, pharmacology

Glycine: PD, pharmacology

Liver: MI, microbiology

Mice

Mice, Inbred BALB C

Random Allocation

Spleen: MI, microbiology

**Triglycerides: PD, pharmacology**

RN 56-40-6 (Glycine); 56-85-9 (Glutamine); 74-79-3 (Arginine)

CN 0 (**Fish Oils**); 0 (Food Additives); 0 (**Triglycerides**)

L109 ANSWER 15 OF 30 MEDLINE on STN

AN 96044639 MEDLINE

DN PubMed ID: 7552772

TI Effects of dietary alpha- and **gamma-linolenic acids** on liver **fatty acids**, lipid metabolism, and survival in **sepsis**.

AU Larsson-Backstrom C; Lindmark L; Svensson L

CS Department of Experimental Biology, Pharmacia AB, Hospital Care, Stockholm, Sweden.

SO Shock (Augusta, Ga.), (1995 Jul) 4 (1) 11-20.  
Journal code: 9421564. ISSN: 1073-2322.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199511

ED Entered STN: 19951227

Last Updated on STN: 19951227

Entered Medline: 19951113

AB The effects of dietary treatment for 3 weeks with soybean oil, linseed- and safflower oil (high **alpha-linolenic acid**

, ALA), or borage oil (high **gamma-linolenic acid**, GLA) on the liver **fatty acid** profile and **lipid** metabolism in fed rats, in normal fasted rats, and **septic** fasted rats, and on survival from **sepsis**, were studied. The results were the following: 1) Dietary ALA increased incorporation of **alpha-linolenic** (18:3w3), **eicosapentaenoic** (20:5w3), and **docosapentaenoic** (22:5w3) acids in neutral **lipids** and phospholipids, and **docosahexaenoic** (22:6w3), **dihomo-gamma-linolenic** (20:3w6), **arachidonic** (20:4w6), stearic (18:0), oleic (18:1w9), and **linoleic** (18:2w6) acids in phospholipids in the livers of fed, fasted, and **septic** fasted rats. Dietary GLA increased all w6 **fatty acids** except 18:2w6, and reduced all w3 **fatty acids** in neutral **lipids** and phospholipids. 2) Dietary ALA increased liver phospholipid content in fasted as well as in **septic** fasted rats and was more potent than GLA in lowering serum cholesterol and liver neutral **lipids**. 3) Dietary ALA counteracted **sepsis**-related changes in liver weight, platelet count, body temperature, prekallikrein, serum glucose, beta-hydroxybutyrate, and free **fatty acids**. 4) Dietary GLA reduced survival from **sepsis**. The results suggest a role for w3 **fatty acids** to balance w6 **fatty acids** in the **septic** state.

CT Check Tags: Comparative Study; Male  
Animals

Cholesterol: BL, blood

Disease Models, Animal

**Fatty Acids: AN, analysis**

\*Liver: DE, drug effects

Liver: ME, metabolism

Phospholipids: CH, chemistry

Phospholipids: ME, metabolism

Rats

Rats, Sprague-Dawley

**Sepsis: DH, diet therapy**

\***Sepsis: ME, metabolism**

**Triglycerides: BL, blood**

\***alpha-Linolenic Acid: PD, pharmacology**

\***gamma-Linolenic Acid: PD, pharmacology**

RN 463-40-1 (**alpha-Linolenic Acid**); 506-26-3 (**gamma-Linolenic Acid**); 57-88-5 (Cholesterol)

CN 0 (**Fatty Acids**); 0 (Phospholipids); 0 (**Triglycerides**)

L109 ANSWER 16 OF 30 MEDLINE on STN

AN 96026991 MEDLINE

DN PubMed ID: 7475985

TI Enteral feeding a structured **lipid** emulsion containing **fish oil** prevents the fatty liver of **sepsis**.

AU Lanza-Jacoby S; Phetteplace H; Tripp R

CS Department of Surgery, Jefferson Medical College, Philadelphia, Pennsylvania 19107, USA.

NC GM31828 (NIGMS)

SO Lipids, (1995 Aug) 30 (8) 707-12.

Journal code: 0060450. ISSN: 0024-4201.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199512

ED Entered STN: 19960124

Last Updated on STN: 19960124

Entered Medline: 19951215

AB **Fish oils** (FO) have been shown to reduce plasma **triglycerides** (TG). In this study we evaluated whether enteral feeding with a structured **lipid** emulsion (SLE) containing FO and **medium-chain triglycerides** (MCT) would prevent the **hypertriglyceridemia** and fatty infiltration of the liver that develops during **sepsis**. For five days, male Lewis rats (275-300 g) were fed intragastrically a nutritionally complete diet containing a SLE or a similar diet with a soybean oil emulsion (SOE) in place of the SLE. On the fifth day, **sepsis** was induced by intravenously injecting  $8 \times 10^7$  live *Escherichia coli* colonies/100 g b.w.; 24 h later the control SLE, **septic** SLE, control SOE, and **septic** SOE rats were sacrificed. Diet, but not treatment, had a significant effect on serum TG and free **fatty acids** (FFA). Feeding the SLE reduced the plasma FFA of the control and **septic** rats by more than 50% in comparison to both control and **septic** rats fed the SOE. Soleus muscle activity of lipoprotein lipase from the **septic** SLE rats was 44% higher than the control SLE rats. Soleus muscle from the **septic** SLE rats also had a twofold greater activity of lipoprotein lipase than the **septic** SOE rats. TG did not accumulate in the livers of the **septic** rats fed SLE when compared to the control SLE rats and the rats fed the SOE. Livers from the **septic** rats fed the SLE had a third of the TG that were present in the livers from the **septic** rats fed the SOE. (ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Male; Support, U.S. Gov't, P.H.S.  
 Animals  
 Cholesterol: BL, blood  
 Emulsions  
 \*Enteral Nutrition  
   \***Fatty Acids**: AD, administration & dosage  
     **Fatty Acids, Nonesterified**: BL, blood  
   Fatty Liver: ET, etiology  
 \*Fatty Liver: PC, prevention & control  
   \***Fish Oils**: AD, administration & dosage  
     **Fish Oils**: TU, therapeutic use  
   **Hypertriglyceridemia**: PC, prevention & control  
 Liver: ME, metabolism  
 Rats  
 Rats, Inbred Lew  
   \***Sepsis**: CO, complications  
 Soybean Oil: AD, administration & dosage  
   \***Triglycerides**: AD, administration & dosage  
     **Triglycerides**: BL, blood  
     **Triglycerides**: ME, metabolism  
 RN 57-88-5 (Cholesterol); 8001-22-7 (Soybean Oil)  
 CN 0 (Emulsions); 0 (**Fatty Acids**); 0 (**Fatty Acids, Nonesterified**); 0 (**Fish Oils**); 0 (**Triglycerides**)

L109 ANSWER 17 OF 30 MEDLINE on STN  
 AN 95178881 MEDLINE  
 DN PubMed ID: 7873917  
 TI Chemically defined structured **lipids** with **omega-3 fatty acids** maintain splanchnic blood flow in a low-dose continuous endotoxin model.  
 AU Pscheidl E; Reisch S; Rugheimer E  
 CS Institut fur Anaesthesiologie, Universitat Erlangen-Nurnberg, FRG.  
 SO Infusionstherapie und Transfusionsmedizin, (1994 Dec) 21 (6) 380-7.  
 Journal code: 9209406. ISSN: 1019-8466.  
 CY Switzerland  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English

FS Priority Journals  
 EM 199504  
 ED Entered STN: 19950419  
 Last Updated on STN: 19950419  
 Entered Medline: 19950406  
 AB BACKGROUND: Disturbances of microcirculation and accompanying alterations of oxygen supply are central pathophysiological events in trauma and **sepsis**. There is evidence that **omega-3 fatty acids** can modulate prostaglandin formation and thereby regional blood flow. The aim of the study was to determine the effects of chemically defined structured **lipids** (SL) with **omega-3 fatty acids** in position sn-2 (MFM) compared to SL with **omega-6 fatty acids** in position sn-2 (MLM) on cardiac output (CO) and splanchnic blood flow in a low-dose endotoxin (E, 1 mg.kgBW-1.day-1) rat model. MATERIALS AND METHODS: 24 male Sprague Dawley rats, divided in 4 groups (n = 6; MLM, MLM+E, MFM, MFM+E) received for 48 h a total parenteral nutrition. CO and regional blood flow were measured with 85strontium-labelled microspheres (16.5 +/- 0.1 microns). RESULTS: There was a slight rise in CO in the E groups compared to the control groups. Application of E resulted in a marked decrease of intestinal perfusion in the MLM-fed animals, whereas the MFM-fed animals showed only a minimal reduction. This decrease of portal blood flow to the liver was accompanied by an elevation of arterial blood flow to the liver. This compensatory increase in arterial liver blood flow was more pronounced in the MFM-fed animals, resulting in a total liver blood flow which was not different from the control group. CONCLUSIONS: The results of this study implicate that 48 h of intravenous feeding with chemically defined SL with an **omega-3 fatty acid** in position sn-2 can significantly influence splanchnic bed perfusion in a low-dose endotoxin rat model. The better splanchnic perfusion may be mediated by a shift in prostaglandin production.  
 CT Check Tags: Comparative Study; Male Animals  
 Digestive System: BS, blood supply  
 \*Endotoxins: BL, blood  
 Energy Metabolism: PH, physiology  
 \*Escherichia coli  
 \*Fat Emulsions, Intravenous  
 \*Fatty Acids, Omega-3: PD, pharmacology  
 Fatty Acids, Omega-6  
 Fatty Acids, Unsaturated: PD, pharmacology  
 Hemodynamic Processes: PH, physiology  
 Kidney: BS, blood supply  
 Liver: BS, blood supply  
 Liver Circulation: PH, physiology  
 Lung: BS, blood supply  
 Muscles: BS, blood supply  
 \*Parenteral Nutrition, Total  
 Rats  
 Rats, Sprague-Dawley  
 Regional Blood Flow: PH, physiology  
 \*Shock, Septic: PP, physiopathology  
 CN 0 (Endotoxins); 0 (Fat Emulsions, Intravenous); 0 (Fatty Acids, Omega-3); 0 (Fatty Acids, Omega-6); 0 (Fatty Acids, Unsaturated)

L109 ANSWER 18 OF 30 MEDLINE on STN  
 AN 95006769 MEDLINE  
 DN PubMed ID: 7922448  
 TI **Omega-3 polyunsaturated fatty acids**  
 : benefit or harm during **sepsis**?

AU Peck M D  
 CS University of Miami/Jackson Memorial Burn Center, University of Miami  
 School of Medicine, FL 33101.  
 SO New horizons (Baltimore, Md.), (1994 May) 2 (2) 230-6. Ref: 68  
 Journal code: 9416195. ISSN: 1063-7389.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199410  
 ED Entered STN: 19941222  
 Last Updated on STN: 19941222  
 Entered Medline: 19941025  
 AB **omega-3 polyunsaturated fatty acids**  
 (PUFAs) are potent modulators of the immune response. Their inclusion in  
 enteral diets may benefit surgical patients recovering from injury or  
 infection. Caution should be used when supplementing **omega-3**  
**PUFAs**, particularly when **fish oil** is used as  
 the source. The long-chain, highly polyunsaturated **fatty**  
**acids in fish oil** are prone to autooxidation,  
 and can potentially damage cells by forming free radicals. In addition,  
**fish oil** may impair the hemostatic response by  
 inhibiting platelet aggregation. Finally, the biochemical and biological  
 end points for the use of **omega-3 PUFAs** have not been  
 clearly established. Thus, although **omega-3 PUFAs**  
 offer tremendous potential as immunomodulators, they also offer potential  
 toxicities, and we must employ them in our clinical practice with this  
 understanding.  
 CT Check Tags: Human  
 Biological Response Modifiers: IM, immunology  
 Biological Response Modifiers: PD, pharmacology  
 \*Biological Response Modifiers: TU, therapeutic use  
 \*Eicosanoids: BI, biosynthesis  
 \*Enteral Nutrition: AE, adverse effects  
 Enteral Nutrition: MT, methods  
**Fatty Acids, Omega-3: IM, immunology**  
**Fatty Acids, Omega-3: PD, pharmacology**  
**\*Fatty Acids, Omega-3: TU, therapeutic use**  
**Fish Oils: IM, immunology**  
**Fish Oils: PD, pharmacology**  
**Fish Oils: TU, therapeutic use**  
 Free Radicals  
 Infection: IM, immunology  
 Infection: ME, metabolism  
 \*Infection: TH, therapy  
**Lipid Peroxidation**  
 Platelet Aggregation: DE, drug effects  
 CN 0 (Biological Response Modifiers); 0 (Eicosanoids); 0 (**Fatty**  
**Acids, Omega-3**); 0 (**Fish**  
**Oils**); 0 (Free Radicals)  
 L109 ANSWER 19 OF 30 MEDLINE on STN  
 AN 94376160 MEDLINE  
 DN PubMed ID: 7916376  
 TI A high alpha-linolenate diet suppresses antigen-induced immunoglobulin E  
 response and anaphylactic **shock** in mice.  
 AU Watanabe S; Sakai N; Yasui Y; Kimura Y; Kobayashi T; Mizutani T; Okuyama H  
 CS Department of Biological Chemistry, Faculty of Pharmaceutical Sciences,  
 Nagoya City University, Japan.  
 SO Journal of nutrition, (1994 Sep) 124 (9) 1566-73.  
 Journal code: 0404243. ISSN: 0022-3166.

CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199410  
 ED Entered STN: 19941031  
 Last Updated on STN: 19980206  
 Entered Medline: 19941020  
 AB Mice were fed for 2 mo diets having ratios of alpha-linolenate [18:3 (n-3)] to linoleate [18:2(n-6)] of < 0.01, 0.36, 1.0 and 3.9. Proportions of safflower seed oil and perilla seed oil were adjusted to obtain these ratios. The dietary alpha-linolenate to linoleate balance was reflected in the proportion of (n-3) and (n-6) highly unsaturated **fatty acids** with 20- and 22-carbon chains in spleen phospholipids, but the ratio did not affect the proportion of T lymphocyte subsets expressing CD4 and CD8 antigens in splenic leukocytes. The immunoglobulin (Ig) G and IgM responses against sheep red blood cells when estimated as plaque-forming cells present in spleen, were not affected significantly by the diets. However, the serum hemagglutinin titer was slightly but significantly higher in the high alpha-linolenate diet group [18:3(n-3)/18:2(n-6) = 3.9] than in the dietary group with 18:3(n-3) to 18:2(n-6) ratios of 0.36 and < 0.01. In contrast, the IgE antibody response against egg albumin, as well as the mortality from anaphylactic **shock** induced by a second challenge with antigen, was significantly lower in the high alpha-linolenate diet group [18:3(n-3)/18:2(n-6) = 3.9] than in the high linoleate diet [18:3(n-3)/18:2(n-6) < 0.01] group. These results, together with the reported suppressive effects of a high alpha-linolenate diet on the formation of **lipid**-derived allergic mediators, support the hypothesis that raising the (n-3) to (n-6) ratios of diets would be effective in reducing the severity of immediate-type allergic hypersensitivity.  
 CT Check Tags: Male; Support, Non-U.S. Gov't  
 \*Anaphylaxis: PC, prevention & control  
 Animals  
 \*Dietary Fats, Unsaturated: AD, administration & dosage  
     **Fatty Acids: AN, analysis**  
     Hemagglutinins: AN, analysis  
 \*Hypersensitivity, Immediate: PC, prevention & control  
 \*Immunoglobulin E: BI, biosynthesis  
     Immunoglobulin G: BI, biosynthesis  
     Immunoglobulin M: BI, biosynthesis  
     **Linoleic Acid**  
     **Linoleic Acids: AD, administration & dosage**  
     Mice  
     Mice, Inbred C3H  
     Mice, Inbred ICR  
     Ovalbumin: IM, immunology  
     Phospholipids: CH, chemistry  
     Spleen: CH, chemistry  
     Spleen: CY, cytology  
     T-Lymphocyte Subsets  
     **\*alpha-Linolenic Acid: AD, administration & dosage**  
 RN 2197-37-7 (Linoleic Acid); 37341-29-0 (Immunoglobulin E);  
 463-40-1 (alpha-Linolenic Acid); 9006-59-1 (Ovalbumin)  
 CN 0 (Dietary Fats, Unsaturated); 0 (**Fatty Acids**); 0 (Hemagglutinins); 0 (Immunoglobulin G); 0 (Immunoglobulin M); 0 (**Linoleic Acids**); 0 (Phospholipids)



AN 94318785 MEDLINE  
 DN PubMed ID: 8043713  
 TI [Fats in parenteral nutrition].  
 Fett in der parenteralen Ernährungstherapie.  
 AU Schricker T; Georgieff M  
 CS Universitätsklinik für Anesthesiologie, Klinikum der Universität Ulm.  
 SO Anesthesiologie, Intensivmedizin, Notfallmedizin, Schmerztherapie : AINS,  
 (1994 May) 29 (3) 137-45. Ref: 45  
 Journal code: 9109478. ISSN: 0939-2661.  
 CY GERMANY: Germany, Federal Republic of  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA German  
 FS Priority Journals  
 EM 199408  
 ED Entered STN: 19940909  
 Last Updated on STN: 19940909  
 Entered Medline: 19940831  
 AB This paper aims at presenting the properties and actions of individual  
**lipids**, as determined via various animal experiments, on the organ  
 systems of the area of the splanchnicus and the lung that are of  
 particular relevance for the intensive-care physician, and to derive  
 possible therapeutic options from these data. Although so far no data are  
 available on newly introduced kinds of fat such as **fish**  
**oil** used in parenteral feeding of critically ill patients, it will  
 be the aim of future therapeutic concepts to arrive at optimal  
 combinations for the individual patient of the pharmacological, energetic  
 and essential properties of the individual classes of **fatty**  
**acids**. Structured **lipids** may possibly be suitable for  
 this task in an ideal manner, the glycerol molecule of which is esterified  
 with **linoleic acid** as an essential constituent of  
 food, a **medium-chain fatty acid** as  
 energy carrier, and an **omega-3 fatty**  
**acid** as immunomodulator. The question as to whether such mixtures  
 can eventually be administered in a not too distant future to parenterally  
 fed patients after trauma and during **septic** condition for their  
 benefit, can be answered only by means of clinical studies that will have  
 to be conducted in years to come.  
 CT Check Tags: Human  
 Animals  
 \*Critical Care  
 Energy Metabolism: PH, physiology  
 English Abstract  
 \*Fat Emulsions, Intravenous: AD, administration & dosage  
 Fat Emulsions, Intravenous: CL, classification  
 Fat Emulsions, Intravenous: ME, metabolism  
 Nutritional Requirements  
 \*Parenteral Nutrition, Total  
 CN 0 (Fat Emulsions, Intravenous)

L109 ANSWER 21 OF 30 MEDLINE on STN  
 AN 93243304 MEDLINE  
 DN PubMed ID: 8480680  
 TI Rapid incorporation of fish or olive oil **fatty acids**  
 into rat hepatic sinusoidal cell phospholipids after continuous enteral  
 feeding during endotoxemia.  
 AU Palombo J D; Bistrian B R; Fechner K D; Blackburn G L; Forse R A  
 CS Department of Surgery, New England Deaconess Hospital, Harvard Medical  
 School, Boston, MA 02215.  
 NC DK31933 (NIDDK)  
 SO American journal of clinical nutrition, (1993 May) 57 (5) 643-9.  
 Journal code: 0376027. ISSN: 0002-9165.

CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199305  
 ED Entered STN: 19930611  
 Last Updated on STN: 19930611  
 Entered Medline: 19930527  
 AB Therapeutic modalities that downregulate macrophage and endothelial production of eicosanoid mediators by displacing membrane **arachidonic acid** (20:4 **omega** 6) may benefit patients at increased risk of **septic** complications. The objective of this study in rats was to assess the incorporation of fish or olive oil **fatty acids** into hepatic Kupffer and endothelial (K&E) cell phospholipids after 4 d of continuous enteral feeding during endotoxemia. Either endotoxin (ETX) (0.5-1 mg-1.day-1) or vehicle was infused intravenously during the last 72 h. Dietary fish and olive oil **fatty acids** were rapidly incorporated into both K&E and plasma phospholipids irrespective of ETX cotreatment. Rats infused with the **fish oil**-enriched diet had a significantly lower relative percent of both K&E **linoleic acid** (18:2 **omega** 6) and 20:4 **omega** 6, whereas rats infused with the olive oil-enriched diet only had a lower relative percent of 18:2 **omega** 6 compared with control rats receiving corn oil. Provision of specific dietary **lipids** by continuous enteral infusion may prove efficacious for the rapid modulation of hepatic sinusoidal cell membrane **fatty acids** under either normal or endotoxemic conditions.  
 CT Check Tags: Male; Support, U.S. Gov't, P.H.S.  
 Animals  
 Dietary Fats: ME, metabolism  
 Endothelium: CY, cytology  
 Endothelium: DE, drug effects  
 Endothelium: ME, metabolism  
 Endotoxins  
 Enteral Nutrition  
 Escherichia coli  
 \*Fatty Acids: ME, metabolism  
 Fatty Acids: PD, pharmacology  
 \*Fish Oils: ME, metabolism  
 Fish Oils: PD, pharmacology  
 Kupffer Cells: DE, drug effects  
 Kupffer Cells: ME, metabolism  
 Liver: CY, cytology  
 Liver: DE, drug effects  
 \*Liver: ME, metabolism  
 \*Phospholipids: ME, metabolism  
 \*Plant Oils: ME, metabolism  
 Plant Oils: PD, pharmacology  
 Rats  
 Rats, Sprague-Dawley  
 Toxemia: DH, diet therapy  
 \*Toxemia: ME, metabolism  
 RN 8001-25-0 (olive oil)  
 CN 0 (Dietary Fats); 0 (Endotoxins); 0 (**Fatty Acids**); 0 (**Fish Oils**); 0 (Phospholipids); 0 (Plant Oils)  
 L109 ANSWER 22 OF 30 MEDLINE on STN  
 AN 92349634 MEDLINE  
 DN PubMed ID: 1640634  
 TI Adaptation to a **fish oil** diet before inducing **sepsis** in rats prevents fatty infiltration of the liver.  
 AU Lanza-Jacoby S; Smythe C; Phetteplace H; Tabares A

CS Department of Surgery, Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA 19107.

NC GM3128 (NIGMS)

SO JPEN. Journal of parenteral and enteral nutrition, (1992 Jul-Aug) 16 (4) 353-8.  
Journal code: 7804134. ISSN: 0148-6071.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199209

ED Entered STN: 19920911  
Last Updated on STN: 19970203  
Entered Medline: 19920903

AB **Hypertriglyceridemia** and fatty liver are common **lipid** abnormalities associated with Gram-negative **sepsis**. **Fish oils** have been shown to have beneficial effects in reducing plasma **triglycerides** (TG). This study was designed to investigate whether **fish oils** would prevent the elevation of plasma TG and the accumulation of liver **lipids** during **sepsis**. One group of rats was fed a 10% menhaden oil diet and the other group was fed a 10% corn oil diet for 14 days. On the 14th day, **sepsis** was induced by injecting the rats with 8 x 10(7) live *Escherichia coli* colonies/100 g of body weight and the rats were fasted for 22 hours. The liver composition of total **lipids** and TG in the **septic** rats prefed the **fish oil** was lower than in the **septic** rats prefed the corn oil. In the rats adapted to the corn oil diet, **lipids** accumulated in the livers of the **septic** rats in comparison with the control rats. Hepatocytes isolated from the **septic** rats adapted to the corn oil diet showed an increased esterification of [1-14C]palmitate into TG and phospholipids than hepatocytes from the control rats. Feeding the **fish oil** diet instead of the corn oil diet before inducing **sepsis** reduced TG, cholesterol, and phospholipid synthesis by 58%, 79%, and 71%, respectively. The rise in TG synthesis in the **septic** rats prefed the corn oil diet was associated with an 89% increase in the activity of phosphatidate phosphohydrolase. There was no significant difference in the activities of glycerol-3-phosphate acyltransferase and phosphatidate phosphohydrolase between control and **septic** rats. (ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S. Animals  
\*Dietary Fats, Unsaturated: TU, therapeutic use  
Escherichia coli Infections  
Esterification  
Fatty Liver: ET, etiology  
Fatty Liver: ME, metabolism  
\*Fatty Liver: PC, prevention & control  
\*Fish Oils: TU, therapeutic use  
Lipids: BL, blood  
Lipoprotein Lipase: ME, metabolism  
Liver: ME, metabolism  
Palmitic Acid  
Palmitic Acids: ME, metabolism  
Phospholipids: ME, metabolism  
Rats  
Rats, Inbred Lew  
\*Septicemia: CO, complications  
Septicemia: ME, metabolism  
Triglycerides: BL, blood  
Triglycerides: ME, metabolism

RN 57-10-3 (Palmitic Acid); 8002-50-4 (Menhaden oil)

CN 0 (Dietary Fats, Unsaturated); 0 (**Fish Oils**); 0 (

**Lipids**); 0 (Palmitic Acids); 0 (Phospholipids); 0 (  
**Triglycerides**); EC 3.1.1.34 (Lipoprotein Lipase)

L109 ANSWER 23 OF 30 MEDLINE on STN  
 AN 92318760 MEDLINE  
 DN PubMed ID: 1619987  
 TI Influence of **omega-3 fatty acids**  
 on splanchnic blood flow and lactate metabolism in an endotoxemic rat  
 model.  
 AU Pscheidl E M; Wan J M; Blackburn G L; Bistrian B R; Istfan N W  
 CS Nutrition-Metabolism Laboratory, New England Deaconess Hospital, Harvard  
 Medical School, Boston, MA 02215.  
 NC DK 40252 (NIDDK)  
 DK 40492 (NIDDK)  
 GM 34074 (NIGMS)  
 SO Metabolism: clinical and experimental, (1992 Jul) 41 (7)  
 698-705.  
 Journal code: 0375267. ISSN: 0026-0495.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199207  
 ED Entered STN: 19920815  
 Last Updated on STN: 19970203  
 Entered Medline: 19920731  
 AB Alteration in regional blood flow is important in the pathogenesis of  
 organ failure during endotoxemia and **sepsis**. In particular,  
 intestinal ischemia is thought to enhance the translocation of bacteria  
 into the systemic circulation. We used radioactive microspheres to  
 measure the influence of two intravenous (IV) dietary fats (vegetable oil  
 containing high levels of **omega-6 fatty**  
**acids**, and **fish oil** containing high levels of  
**omega-3 fatty acids**) on regional  
 blood flow during low-dose Escherichia coli endotoxin infusion (0.1 mg/100  
 g body weight [BW]) in a rat model. Despite absence of changes in the  
 cardiac output, blood flow rates to the small and large intestines,  
 stomach, and pancreas, and also to the skin and skeletal muscle were  
 significantly reduced after 18 hours of endotoxin infusion in the rats fed  
 standard vegetable oil. Short-term IV feeding during a period of 40 hours  
 with an isonitrogenous, isocaloric nutrient solution containing  
**fish oil** as the only **lipid** source normalized  
 intestinal perfusion and increased blood flow to the liver and spleen.  
 Low-dose endotoxin infusion also resulted in significant increases in  
 glucose, lactate, and pyruvate concentrations. In comparison to standard  
 vegetable fat emulsion, **fish oil** significantly reduced  
 these parameters. A second experiment was conducted to measure lactate  
 kinetics. Based on the dilution of U-14C-lactate, **fish**  
**oil** feeding was associated with higher lactate clearance than  
 standard vegetable oil feeding during the endotoxin infusion. We conclude  
 that short-term IV feeding with **fish oil** improves  
 intestinal perfusion and portal blood flow, improves glucose tolerance,  
 and increases lactate clearance in a low-dose endotoxin rat model.  
 CT Check Tags: Male; Support, U.S. Gov't, P.H.S.  
 Animals  
 Cardiac Output: DE, drug effects  
 \*Endotoxins: TO, toxicity  
 \*Fatty Acids, Omega-3: PD, pharmacology  
 Fish Oils: PD, pharmacology  
 Glucose: ME, metabolism  
 \*Lactates: ME, metabolism  
 Lactic Acid  
 Rats

Rats, Inbred Strains

\*Splanchnic Circulation: DE, drug effects

Thromboxane A2: PH, physiology

\*Toxemia: PP, physiopathology

RN 50-21-5 (Lactic Acid); 50-99-7 (Glucose); 57576-52-0 (Thromboxane A2)

CN 0 (Endotoxins); 0 (**Fatty Acids, Omega-3**); 0 (**Fish Oils**); 0 (Lactates)

L109 ANSWER 24 OF 30 MEDLINE on STN

AN 92048543 MEDLINE

DN PubMed ID: 1943744

TI Long-term feeding with structured **lipid** composed of **medium-chain** and **N-3 fatty acids** ameliorates endotoxic **shock** in guinea pigs.

AU Teo T C; Selleck K M; Wan J M; Pomposelli J J; Babayan V K; Blackburn G L; Bistrrian B R

CS Department of Surgery, Aberdeen Royal Infirmary, Foresterhill, Scotland.

NC DK 40252 (NIDDK)

DK 41128 (NIDDK)

SO Metabolism: clinical and experimental, (1991 Nov) 40 (11) 1152-9.

Journal code: 0375267. ISSN: 0026-0495.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199112

ED Entered STN: 19920124

Last Updated on STN: 19970203

Entered Medline: 19911212

AB The metabolic and physiologic responses to 7-hour endotoxin infusion (5.0 mg/kg h) were evaluated in guinea pigs following 6 weeks of dietary enrichment with diets containing either chemically structured **lipid** (SL) composed of **medium-chain triglycerides** (MCT) and long-chain **triglycerides** (LCT) in the form of **N-3 polyunsaturated fatty acids** (PUFAs), or safflower oil (SO), which is high in **N-6 fatty acids**. Plasma phospholipid **fatty acid** profiles, arterial blood pH, PCO<sub>2</sub>, PO<sub>2</sub>, HCO<sub>3</sub>, lactate, blood pressure, oxygen consumption, and energy expenditure were examined. Plasma phospholipid **fatty acids** profiles reflected dietary intake with SL-fed animals demonstrating a significantly higher **N-3 to N-6 fatty acid** ratio compared with SO-fed animals. SL-fed animals responded to endotoxemia with a mild metabolic acidosis with respiratory compensation, which was associated with moderate lactatemia (3 mmol/L). SO-fed animals developed a severe metabolic acidosis with acidemia and respiratory compensation, which was associated with hyperlactatemia (8 mmol/L, P less than .05 v SL). No differences were observed in blood pressure, oxygen consumption, energy expenditure, or respiratory quotient during endotoxemia between dietary groups compared with controls. We conclude that diets enriched with structured **lipid** composed of **medium-chain and N-3 fatty acids** can attenuate the sequelae of endotoxemia.

CT Check Tags: Male; Support, U.S. Gov't, P.H.S.

Animals

Blood Pressure

Calorimetry, Indirect

\*Dietary Fats: PD, pharmacology

**Fatty Acids: BL, blood**

\***Fatty Acids: PD, pharmacology**

Guinea Pigs

Lactates: BL, blood

Lactic Acid

**Lipids: CH, chemistry**

**\*Lipids: PD, pharmacology**

Phospholipids: BL, blood

**Shock, Septic: ME, metabolism**

**\*Shock, Septic: PP, physiopathology**

Time Factors

RN 50-21-5 (Lactic Acid)

CN 0 (Dietary Fats); 0 (**Fatty Acids**); 0 (Lactates); 0 (**Lipids**); 0 (Phospholipids)

L109 ANSWER 25 OF 30 MEDLINE on STN

AN 91318319 MEDLINE

DN PubMed ID: 1861166

TI High dietary **linoleic acid** affects the **fatty acid** compositions of individual phospholipids from tissues of Atlantic salmon (*Salmo salar*): association with stress susceptibility and cardiac lesion.

AU Bell J G; McVicar A H; Park M T; Sargent J R

CS Unit of Aquatic Biochemistry, School of Natural Sciences, University of Stirling, Scotland.

SO Journal of nutrition, (1991 Aug) 121 (8) 1163-72.

Journal code: 0404243. ISSN: 0022-3166.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199108

ED Entered STN: 19910922

Last Updated on STN: 19980206

Entered Medline: 19910830

AB For 16 wk Atlantic salmon (*Salmo salar*) post-smolts were fed practical-type diets that contained either **fish oil** (FO) or sunflower oil (SO) as the **lipid** component. Both diets contained adequate (n-3) polyunsaturated **fatty acids** (PUFA). All the phospholipids of heart and liver from SO-fed fish had increased levels of 18:2(n-6), 20:2(n-6) and 20:3(n-6); phosphatidyl choline (PC) and phosphatidyl ethanolamine (PE) also had increased 20:4(n-6). There was a general decrease in 20:5(n-3) in the phospholipids, reflected in an increase in the 20:4(n-6)/20:5(n-3) ratio, especially in PC and PE. The **fatty acid** compositions of phospholipids from brain and retina were much less affected by dietary linoleate than those of heart and liver. Fish fed SO developed severe heart lesions that caused thinning of the ventricular wall and muscle necrosis. The fish fed SO also were susceptible to a transportation-induced **shock** syndrome that caused 30% mortality. These results establish that a diet with a low (n-3)/(n-6) ratio can cause changes in **fatty acid** metabolism that are deleterious to the health of salmonid fish, especially when subjected to stress.

CT Animals

Brain: DE, drug effects

Brain: ME, metabolism

Dietary Fats, Unsaturated: AD, administration & dosage

**\*Dietary Fats, Unsaturated: PD, pharmacology**

**\*Fatty Acids: ME, metabolism**

**Fish Oils: AD, administration & dosage**

**Fish Oils: PD, pharmacology**

Heart: DE, drug effects

Heart Diseases: ET, etiology

Heart Diseases: PA, pathology

\*Heart Diseases: VE, veterinary

Linoleic Acid

Linoleic Acids: AD, administration & dosage

Linoleic Acids: AE, adverse effects

\*Linoleic Acids: PD, pharmacology

Liver: DE, drug effects

Liver: ME, metabolism

Myocardium: ME, metabolism

Myocardium: PA, pathology

Necrosis

Phosphatidylcholines: ME, metabolism

Phosphatidylethanolamines: ME, metabolism

\*Phospholipids: ME, metabolism

Plant Oils: AD, administration & dosage

Plant Oils: PD, pharmacology

Retina: DE, drug effects

Retina: ME, metabolism

\*Salmon

Stress: ET, etiology

\*Stress: VE, veterinary

RN 2197-37-7 (Linoleic Acid); 8001-21-6 (sunflower seed oil)

CN 0 (Dietary Fats, Unsaturated); 0 (Fatty Acids); 0 (Fish Oils); 0 (Linoleic Acids); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phospholipids); 0 (Plant Oils)

L109 ANSWER 26 OF 30 MEDLINE on STN

AN 91269439 MEDLINE

DN PubMed ID: 1904949

TI Effects of a fish oil diet on pigs' cardiopulmonary response to bacteremia.

AU Murray M J; Svingen B A; Holman R T; Yaksh T L

CS Critical Care Service, Mayo Clinic, Rochester, MN 55905.

SO JPEN. Journal of parenteral and enteral nutrition, (1991 Mar-Apr) 15 (2) 152-8.

Journal code: 7804134. ISSN: 0148-6071.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199107

ED Entered STN: 19910811

Last Updated on STN: 19910811

Entered Medline: 19910722

AB Since an omega 3 fatty acid (FA)

diet may have beneficial effects in inflammatory processes, we tested the hypothesis that the physiologic response to sepsis could be modified by altering the eicosanoid precursor pool via an omega 3 FA diet. Two groups (n = 8) of pigs were prefed for 8 days either an omega 3 FA or an omega 6

FA diet (Weaner Pig Feed with either menhaden or corn oil to produce a eucaloric feed with 15% fat) and then injected with live Escherichia coli. The omega 3 FA diet increased the concentration of eicosapentanoic acid (EPA, 20:5 omega 3) in plasma lipids, and increased the ratio of EPA to arachidonic acid (AA, 20:4 omega 6) in platelets from 1:20

to 1:1 over the 8 days. Following the injection of bacteria, there was a fall in PaO2 and blood pressure that was attenuated (p less than 0.05) by the omega 3 FA diet. The omega 3

FA diet, compared to the omega 6 FA diet, also attenuated the rise in thromboxane B2 (3.0 +/- 1.1 vs 12.9 +/- 5.7 ng/mL) and 6 keto-PGF1 alpha (0.8 +/- 0.5 vs 1.7 +/- 1.1 ng/mL) associated with bacteremia. We conclude that dietary omega 3 FA

attenuated the physiologic response to **sepsis**, possibly by modifying **arachidonic acid** metabolism.

CT Check Tags: Male

6-Ketoprostaglandin F1 alpha: BL, blood

Animals

**Arachidonic Acid**

**Arachidonic Acids: BL, blood**

Blood Platelets: ME, metabolism

Blood Pressure

\*Cardiovascular System: PP, physiopathology

\*Dietary Fats, Unsaturated: TU, therapeutic use

**Eicosapentaenoic Acid: BL, blood**

Escherichia coli Infections: CO, complications

\*Escherichia coli Infections: PP, physiopathology

**\*Fish Oils: TU, therapeutic use**

**Lipids: BL, blood**

\*Lung: PP, physiopathology

Norepinephrine: BL, blood

Oxygen: BL, blood

Pulmonary Edema: ET, etiology

**Septicemia: CO, complications**

**Septicemia: DH, diet therapy**

**\*Septicemia: PP, physiopathology**

Swine

Thromboxane B2: BL, blood

RN 1553-41-9 (**Eicosapentaenoic Acid**); 506-32-1 (**Arachidonic Acid**); 51-41-2 (Norepinephrine); 54397-85-2 (Thromboxane B2); 58962-34-8 (6-Ketoprostaglandin F1 alpha); 7782-44-7 (Oxygen)

CN 0 (**Arachidonic Acids**); 0 (Dietary Fats, Unsaturated); 0 (**Fish Oils**); 0 (**Lipids**)

L109 ANSWER 27 OF 30 MEDLINE on STN

AN 91187922 MEDLINE

DN PubMed ID: 2011612

TI Free radical generation, **lipid** peroxidation and essential **fatty acids** in patients with **septicemia**.

AU Prabha P S; Das U N; Ramesh G; Kumar K V; Kamalakara V

CS Department of Medicine, Nizam's Institute of Medical Sciences, Hyderabad, India.

SO Prostaglandins, leukotrienes, and essential fatty acids, (1991 Jan) 42 (1) 61-5.

Journal code: 8802730. ISSN: 0952-3278.

CY SCOTLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199105

ED Entered STN: 19910526

Last Updated on STN: 19910526

Entered Medline: 19910506

AB Infections due to gram-negative bacteria and other organisms can lead to **septicemia** and **shock** in some patients. Endotoxins, which cause these pathophysiological events, stimulate macrophages to elaborate tumor necrosis factor and other lymphokines. These lymphokines can augment free radical generation by polymorphonuclear leukocytes, macrophages and other cells, which may ultimately produce respiratory distress syndrome, multiorgan failure and irreversible **shock** seen in **septicemia**. This is supported by our results presented here that there is indeed an increase in free radical generation and **lipid** peroxidation in patients with **septicemia**. In addition, analysis of plasma **lipid** profile in these patients showed that gamma-linolenic, dihomogamma-linolenic and **arachidonic acids** of n-6 series and



alpha-linolenic and eicosapentaenoic acids of the n-3 series are decreased in their plasma phospholipid fraction. These results suggest that free radicals, lipid peroxides, and alteration in essential fatty acid metabolism may have a role in the pathogenesis of septicemia.

CT Check Tags: Human; Support, Non-U.S. Gov't

Adolescent

Adult

Aged

Animals

Dogs

\*Fatty Acids: BL, blood

Free Radicals

\*Gram-Negative Bacteria: ME, metabolism

\*Lipid Peroxidation

Lipid Peroxides: BL, blood

Middle Aged

Neutrophils: DE, drug effects

\*Neutrophils: ME, metabolism

Phospholipids: BL, blood

\*Septicemia: ME, metabolism

Septicemia: MI, microbiology

Shock

CN 0 (Fatty Acids); 0 (Free Radicals); 0 (Lipid Peroxides); 0 (Phospholipids)

L109 ANSWER 28 OF 30 MEDLINE on STN

AN 91128027 MEDLINE

DN PubMed ID: 1992944

TI Diets enriched with N-3 fatty acids

ameliorate lactic acidosis by improving endotoxin-induced tissue hypoperfusion in guinea pigs.

AU Pomposelli J J; Flores E A; Blackburn G L; Zeisel S H; Bistrian B R

CS Cancer Research Institute, New England Deaconess Hospital, Boston, MA 02215.

NC AM31933 (NIADDK)

GM30632 (NIGMS)

SO Annals of surgery, (1991 Feb) 213 (2) 166-76.

Journal code: 0372354. ISSN: 0003-4932.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 199103

ED Entered STN: 19910405

Last Updated on STN: 19910405

Entered Medline: 19910314

AB The effect of 6 weeks dietary lipid manipulation on the acute physiologic response to 7-hour continuous endotoxin infusion in guinea pigs was examined. One diet was enriched with N-3 fatty acids, whereas the other contained N-6 fatty acids, primarily linoleic acid. Animals fed N-6 fatty acids developed significant lactic acidemia, microvascular muscle hypoperfusion, and pulmonary infiltrates in response to endotoxin infusion. N-3 fatty acid-fed animals demonstrated improved lactate levels, microvascular muscle perfusion, and lung morphology compared to N-6 fatty acid-fed animals after endotoxin infusion. There was no significant change in cardiac output, PaO<sub>2</sub>, or mean arterial blood pressure at the end of the endotoxin infusion in either group. Pretreatment with indomethacin, or BM 13505, a specific thromboxane A<sub>2</sub>

receptor blocker, ameliorated the development of metabolic acidosis in **N-6 fatty acid**-fed animals, demonstrating a role for prostanoids in the sequelae of endotoxemia. The ability of dietary pretreatment with **N-3 fatty acids** to influence favorably the physiologic response to endotoxin represents a novel nutrient-metabolic interaction with potential therapeutic implications.

CT Check Tags: Male; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.  
 \*Acidosis, Lactic: BL, blood  
 Acidosis, Lactic: ET, etiology  
 Acidosis, Lactic: TH, therapy  
 Animals  
 Dietary Fats: AD, administration & dosage  
 Dietary Fats: PD, pharmacology  
   **Fatty Acids, Omega-3: AD, administration & dosage**  
   **Fatty Acids, Unsaturated: AD, administration & dosage**  
   **Fish Oils: AD, administration & dosage**  
 Guinea Pigs  
 \*Hemodynamic Processes: DE, drug effects  
 Indomethacin: PD, pharmacology  
 Lung: PA, pathology  
 Muscles: BS, blood supply  
 Phenylacetates: AD, administration & dosage  
 Regional Blood Flow: DE, drug effects  
 Safflower Oil: AD, administration & dosage  
   **Shock, Septic: CO, complications**  
   **Shock, Septic: PA, pathology**  
   **\*Shock, Septic: PP, physiopathology**  
 Skin: BS, blood supply  
 Sulfonamides: AD, administration & dosage  
 Thromboxanes: AI, antagonists & inhibitors  
 RN 105218-03-9 (daltroban); 53-86-1 (Indomethacin); 8001-23-8 (Safflower Oil)  
 CN 0 (Dietary Fats); 0 (**Fatty Acids, Omega-3**); 0 (**Fatty Acids, Unsaturated**); 0 (**Fish Oils**); 0 (Phenylacetates); 0 (Sulfonamides); 0 (Thromboxanes)

L109 ANSWER 29 OF 30 MEDLINE on STN

AN 89237146 MEDLINE

DN PubMed ID: 2541281

TI Modulation of Kupffer cell membrane phospholipid function by **n-3 polyunsaturated fatty acids**.

AU Bankey P E; Billiar T R; Wang W Y; Carlson A; Holman R T; Cerra F B

CS Department of Surgery, University of Minnesota, Minneapolis.

SO Journal of surgical research, (1989 May) 46 (5) 439-44.

Journal code: 0376340. ISSN: 0022-4804.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 198906

ED Entered STN: 19900306

Last Updated on STN: 19900306

Entered Medline: 19890614

AB Dietary **n-3 polyunsaturated fatty**

**acids** (PUFAs) have been reported to improve clinical outcome in a number of inflammatory diseases including burns and **sepsis**. One mechanism contributing to the anti-inflammatory effect is the incorporation of **n-3 PUFAs** into membrane phospholipids which decreases macrophage eicosanoid production. We hypothesize that an additional mechanism for their effects is an alteration of membrane signal transduction that decreases macrophage responsiveness to inflammatory stimuli. Kupffer cells, the fixed macrophages of the liver, were obtained

from rats pair fed diets for 6 weeks with 15% of calories supplied as menhaden (high n-3), corn (control), or safflower (high n-6) oils. The effects of the dietary oils on Kupffer cell membrane signal transduction and eicosanoid production were assessed by measuring inositol phospholipid (PI) metabolism, intracellular calcium responses, and prostaglandin E2 (PGE2) production to the inflammatory signals endotoxin (LPS) and platelet activating factor (PAF). The menhaden oil diet resulted in significant incorporation of n-3 PUFAs into total cellular PUFAs compared to corn and safflower oil. (total n-3 PUFAs, 28.1% menhaden vs 2.1% corn vs 1.2% safflower, P less than 0.03). This incorporation altered signal transduction of PAF as both PI turnover (65% +/- 10% of corn oil) and calcium response (0.6-fold vs 5.0-fold for corn oil) were significantly reduced in the menhaden oil group. (P less than 0.05) The menhaden oil diet also reduced significantly PGE2 production in response to PAF and LPS (corn, 348 +/- 23 pg/ml; menhaden, 48 +/- 6 pg/ml, P less than 0.01). We conclude that, in addition to modulating eicosanoid production, n-3 PUFAs can also alter macrophage membrane signal transduction. (ABSTRACT TRUNCATED AT 250 WORDS)

CT Check Tags: Male

Animals

Calcium: ME, metabolism

Calcium Channels: ME, metabolism

Corn Oil: ME, metabolism

\*Dietary Fats: ME, metabolism

Dinoprostone: ME, metabolism

\*Fatty Acids, Unsaturated: ME, metabolism

Inflammation: ME, metabolism

\*Kupffer Cells: ME, metabolism

\*Membrane Lipids: ME, metabolism

\*Phospholipids: ME, metabolism

Rats

Rats, Inbred Strains

Safflower Oil: ME, metabolism

RN 363-24-6 (Dinoprostone); 7440-70-2 (Calcium); 8001-23-8 (Safflower Oil); 8001-30-7 (Corn Oil)

CN 0 (Calcium Channels); 0 (Dietary Fats); 0 (Fatty Acids, Unsaturated); 0 (Membrane Lipids); 0 (Phospholipids)

L109 ANSWER 30 OF 30 MEDLINE on STN

AN 88291006 MEDLINE

DN PubMed ID: 3041642

TI Fatty acid intake and Kupffer cell function:

fish oil alters eicosanoid and monokine production to endotoxin stimulation.

AU Billiar T R; Bankey P E; Svingen B A; Curran R D; West M A; Holman R T; Simmons R L; Cerra F B

CS Department of Surgery, University of Minnesota, Minneapolis.

SO Surgery, (1988 Aug) 104 (2) 343-9.

Journal code: 0417347. ISSN: 0039-6060.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 198809

ED Entered STN: 19900308

Last Updated on STN: 19900308

Entered Medline: 19880902

AB Diets high in n-3 fatty acids

appear to have an anti-inflammatory effect, which is thought to be due to decreased macrophage prostaglandin (PG) and thromboxane (Tx) production after incorporation of these fatty acids into cell membrane phospholipids. The effect of n-3

**fatty acids** incorporation on macrophage monokine release in response to **septic** stimuli is not well established. Kupffer cells, the fixed macrophages of the liver, were obtained from rats fed diets with fat sources derived from corn oil (CO, control), **fish oil** (FO, high in **n-3 fatty acids**), or safflower oil (SO, high in **n-6 fatty acids**) for 2 or 6 weeks. After exposure to bacterial lipopolysaccharide, Kupffer cells from rats fed FO for 2 or 6 weeks produced less PG and Tx than Kupffer cells from rats fed CO or SO. After 2 weeks of defined diets, interleukin-1 (IL-1) and tumor necrosis factor release were not affected by dietary fat source. In contrast, after 6 weeks of feeding, Kupffer cells from both the FO and the SO groups released less IL-1 and tumor necrosis factor when triggered by lipopolysaccharide than Kupffer's cells from animals fed the control diet that contained CO. These data suggest that altered monokine release from macrophages may contribute to the anti-inflammatory effect of diets high in **n-3 fatty acids**. Also shown in our results is that prolonged changes in membrane phospholipid content induced by dietary fat source can influence not only PG and Tx production but monokine release as well.

CT Check Tags: Comparative Study

Animals

\*Dietary Fats: PD, pharmacology

Escherichia coli

**Fatty Acids, Unsaturated: AD, administration & dosage**

**\*Fatty Acids, Unsaturated: PD, pharmacology**

**Fish Oils: PD, pharmacology**

Interleukin-1: BI, biosynthesis

Kupffer Cells: IM, immunology

\*Kupffer Cells: PH, physiology

Lipopolysaccharides: PD, pharmacology

Lymphokines: IM, immunology

**Membrane Lipids: PH, physiology**

Prostaglandins: BI, biosynthesis

Rats

Rats, Inbred Strains

Thromboxanes: BI, biosynthesis

Tumor Necrosis Factor: BI, biosynthesis

CN 0 (Dietary Fats); 0 (**Fatty Acids, Unsaturated**); 0 (**Fish Oils**); 0 (Interleukin-1); 0 (Lipopolysaccharides); 0 (Lymphokines); 0 (**Membrane Lipids**); 0 (Prostaglandins); 0 (Thromboxanes); 0 (Tumor Necrosis Factor)

=> => fil wpix

FILE 'WPIX' ENTERED AT 11:22:07 ON 31 AUG 2004

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FILE LAST UPDATED: 26 AUG 2004 <20040826/UP>

MOST RECENT DERWENT UPDATE: 200455 <200455/DW>

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HIT STRUCTURES WITHIN THE BIBLIOGRAPHIC DOCUMENT <<<

=> d all abeq tech abex tot

L142 ANSWER 1 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN  
AN 2003-416447 [39] WPIX  
CR 1999-527413 [44]  
DNC C2003-110187  
TI Oil-in-water emulsion useful for treating e.g. cancer comprises  
**gamma linolenic acid**, **eicosapentaenoic acid** as  
delta-desaturase inhibitor, emulsifying agent or emulsion stabilizer and  
water.  
DC A96 B05 D13  
IN CHILTON, F H; KOUMENIS, I L; SURETTE, M E; TRAMPOSCH, K  
PA (CHIL-I) CHILTON F H; (KOU-M-I) KOUMENIS I L; (SURE-I) SURETTE M E;  
(PILO-N) PILOT THERAPEUTICS INC  
CYC 102  
PI US 2002188024 A1 20021212 (200339)\* 50 A61K031-202  
WO 2003063793 A2 20030807 (200361) EN A61K000-00  
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS  
LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT  
RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA  
ZM ZW  
AU 2003208920 A1 20030902 (200422) A61K031-202  
ADT US 2002188024 A1 CIP of US 1998-28256 19980223, CIP of WO  
1999-US3120 19990212, CIP of US 2000-644380 20000823, US 2002-66334  
20020131; WO 2003063793 A2 WO 2003-US2954 20030131; AU 2003208920 A1 AU  
2003-208920 20030131  
FDT US 2002188024 A1 CIP of US 6107334; AU 2003208920 A1 Based on WO  
2003063793  
PRAI US 2002-66334 20020131; US 1998-28256  
19980223; WO 1999-US3120 19990212; US  
2000-644380 20000823  
IC ICM A61K000-00; A61K031-202  
AB US2002188024 A UPAB: 20040331  
NOVELTY - Oil-in-water emulsion comprises **gamma -  
linolenic acid** (GLA) (0.5 - 3, pediatric 0.2 - 3 g),  
**eicosapentaenoic acid** (EPA) as a Delta 5-desaturase inhibitor (0.1  
- 3, pediatric 0.02 - 3 g), an emulsifying agent or emulsion stabilizer  
and water, where the bioavailability of the emulsified GLA and EPA is  
greater than that of GLA and EPA administered in gel capsule form.  
ACTIVITY - Antiasthmatic; Antiallergic; **Antiinflammatory**;  
Antipsoriatic; Cardiant; Nephrotropic; Gastrointestinal-Gen.;  
Antiarthritic; Cytostatic; Dermatological; Immunosuppressive; Neuroleptic;  
Antidepressant; Antibacterial; Vasotropic; Nootropic; Neuroprotective;  
Antiartherosclerotic; Gynecological; Analgesic; Tocolytic; Antigout;  
Antiulcer; Antithyroid; Muscular-Gen.; Osteopathic; Antirheumatic;  
Uropathic; Ophthalmological; Tranquilizer.  
MECHANISM OF ACTION - Delta 5-desaturase Inhibitor; Serum  
**Arachidonic Acid Increase Inhibitor**.  
In tests to evaluate the influence of the combination of  
**gamma -linolenic acid** (GLA) and **eicosapentaenoic  
acid** (EPA) on the **fatty acid** composition of serum and  
neutrophil **lipids**, three subjects on control diet (25% fat) were

supplemented with a combination of EPA (1.5 g/day) and GLA (3.0 g/day) for three weeks. GLA when administered alone showed accumulation of **arachidonic acid (AA)** and **dihomo- gamma - linolenic acid (DGLA)** in serum and polymorphonuclear (PMN) cells. The AA/DGLA accumulation (micro mol/5 million PMN) was found to be 0.9/0.125 (at 0 weeks), 1/0.25 (after 4 weeks), 1.125/0.25 (after 8 weeks), and 1/0.24 (after 12 weeks), respectively. The results showed that GLA alone induced marked increase in serum AA in both short (3 weeks) and the long term (12 weeks). However, the combination of GLA and EPA did not cause an increase in the serum AA levels. The **fatty acids** concentration in the serum of AA/GLA/DGLA/EPA (micro mol/l serum) was 350/20/100/20 (at 0 weeks), 350/25/105/100 (after 1 week), 450/50/250/260 (after 2 weeks) 400/40/160/100 (after 3 weeks), and 450/50/100/40 (after washout). The results showed that blocking of Delta 5-desaturase in humans provides a means to supplement humans with high levels of GLA without concomitant increase in serum AA levels.

USE - The emulsion is useful for treating a **lipid-mediated** disorder having an **arachidonic acid** metabolite component e.g. asthma, allergic rhinitis, allergic rhinoconjunctivitis, psoriasis, acute myocardial infarction, glomerulonephritis, Crohn's disease, **inflammatory** bowel disease, arthritis, breast cancer, colon cancer, prostate cancer, squamous cell carcinoma, intestinal cancer, ovarian cancer, uterine cancer, testicular cancer, autoimmune disease, systemic lupus erythematosus, schizophrenia, depression, immunoglobulin A nephropathy, **sepsis**, toxic **shock**, organ failure, organ transplant, coronary angioplasty, risk reduction for Alzheimer's disease, cystic fibrosis, atherosclerosis, atopic dermatitis, menstrual discomfort, cyclic breast pain, premature labor, early parturition, gout, venous leg ulcers, chronic urticaria, thyroiditis, primary dysmenorrhea, endometriosis, Lyme disease, muscle wasting, ankylosing spondylitis, carpal tunnel syndrome, childhood or juvenile arthritis, chronic back injury, fibromyalgia, infectious arthritis, osteoarthritis, osteoporosis, Paget's disease, polymyalgia rheumatica, polymyositis, dermatomyositis, pseudogout, psoriatic arthritis, Raynaud's syndrome, reactive arthritis, Reiter's syndrome, repetitive stress injury, rheumatoid arthritis, scleroderma and Sjogrens syndrome (all claimed). Also useful for controlling or reducing the symptoms of **inflammation**, and as a dietary and pediatric formulations.

ADVANTAGE - The bioavailability of emulsified GLA and EPA is greater than the bioavailability of GLA and EPA administered in gel form. The combination of GLA and Delta 5-desaturase inhibitor EPA prevents the increase in serum **arachidonic acid** level upon GLA administration. Additionally **omega -3-AA** is provided and the build up of **omega -3-AA** in neutrophils results in further inhibition of the serum Delta 5-desaturation of DGLA in hepatocytes, resulting in further inhibition of serum AA accumulation.

Dwg.0/25

FS CPI

FA AB; DCN

MC CPI: A12-V01; B03-F; **B04-B01B**; B04-B01C1; B04-C02A2; B04-C02B; B04-C02D; B04-N02; B05-B01P; B07-A02A; B10-C04E; B12-M03; B12-M06; B14-A01; B14-C01; B14-C02; B14-C03; B14-C09; B14-D03; B14-E08; B14-E10C; B14-E11; B14-F01B; B14-F02; B14-F04; B14-F07; B14-G02A; B14-G02C; B14-G02D; B14-H01; B14-J01A1; B14-J01A4; B14-J01B3; B14-J01B4; B14-J05; B14-K01; B14-N01; B14-N03; B14-N04; B14-N07; B14-N10; B14-N11; B14-N12; B14-N14; B14-N16; B14-N17; B14-N18; B14-P03; **B14-S06**; B14-S09; D03-A; D03-H01A; D03-H01C; D03-H01E; D03-H01N; **D03-H01T2**; D03-H02

TECH UPTX: 20030619

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Formulation: The formulation additionally comprises at least one flavoring agent, sweetening agent, coloring agent or a preservative. Preferred Method: The administration of the emulsion alters the synthesis

of at least one **arachidonic acid (AA)** metabolite.

Preferred Components: The AA metabolite is at least one of leukotrienes (LT), prostaglandins (PG) or lipoxins (LX) (preferably 5-hydroperoxy-eicosatetraenoic acid, 5-hydroxyeicosatetraenoic acid (5-HETE), 5-HETE-lactone, leukotriene A4 (LTA4), 5(S),6(S)-dihydroxyeicosatetraenoic acid (DIHETE), 5(S),6(R)-DIHETE, LTB4, lipoxin A4 (LXA4), LTC4, 12(R,S),6-trans-LTB4, LTF4, LTD4, LTE4, 20-OH-LTE4, 20-COOH-LTE4, 18-COOH-LTE4, 16-COOH-LTE3, 14-COOH-LTE3, LTE4-Nac, prostaglandin G2 (PGG2), PGH2, PGD2, 13,14-dihydro-15-keto-PGD2, 9-alpha,11-beta-PGF2, PGJ2, DELTA12-PGJ2, PGE2, 9alpha,11alpha-PGF2, PGA2, PGB2, 19-OH-PGE2, 15-keto-PGE2, 13,14-dihydro-15-keto-PGE2, PGE-M, PGF2alpha, 15-keto-PGF2alpha, 13,14-dihydro-15-keto-PGF2alpha, PGF-M, lipoxin A4, lipoxin B4, 15-epi-lipoxin A4 or 15-epi-lipoxin A5; especially leukotriene; particularly LTB4).

TECHNOLOGY FOCUS - POLYMERS - Preferred Components: The emulsifying agent or emulsion stabilizer is xanthan gum, guar gum, pectin, carob seed gum (locust-bean gum), tragacanth gum, methylcellulose, starch, modified starch, carboxymethylcellulose, gum Arabic or gelatin, **phospholipid**, lecithin, alginates or carrageenan.

ABEX UPTX: 20030619

ADMINISTRATION - The emulsion is administered in a unit dosage form, providing a daily dosage of EPA and GLA (claimed). The emulsion is administered orally. The dosage comprises GLA 1 - 15 (preferably 1.5 - 3) g; EPA 0.1 - 10 (preferably 0.5 - 3) g, and optionally stearidonic acid 0.1 - 15 (preferably 3 - 5) g, administered 1 - 4 times daily. The dosage of GLA is 0.2 - 3, and of EPA is 0.02 - 3, when used as a pediatric formulation.

EXAMPLE - A dietary fatty acid emulsion was prepared as follows. A stabilized emulsion comprised (g): borage oil (21), marine oil (16.5), lecithin (0.5), flavor and flavor masking agent (2), colorant (0.05), ascorbyl palmitate (0.2), sorbic acid (0.16), sucrose (25), xanthan gum (0.3), water (29.29) and glycerin (5). The emulsion prepared was preferably packaged in an oxygen-free environment in single daily dosage packages made of oxygen impermeable materials, such as foil-lined pouches. A dosage of 20 g/day was recommended, along with **gamma-linolenic acid** (1.5 g) and **eicosapentaenoic acid** (1.0 g).

L142 ANSWER 2 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 2001-281492 [29] WPIX

DNC C2001-085515

TI Composition for use as a medicament, food or nutritive product, for the treatment of **sepsis** or **inflammatory shock**, comprises at least one **lipid**.

DC B05 C03 D13

IN BREUILLE, D; CROZIER-WILLI, G; DUTOT, G; FINOT, P; OBLED, C; RICHELLE, M; ROESSLE, C; TURINI, M

PA (INRG) INRA INST NAT RECH AGRONOMIQUE; (NEST) SOC PROD NESTLE SA

CYC 95

PI WO 2001019356 A2 20010322 (200129)\* EN 29 A61K031-00

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM  
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC  
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE  
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

EP 1090636 A1 20010411 (200129) EN A61K031-23

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI

AU 2000068430 A 20010417 (200140) A61K031-00

BR 2000013958 A 20020514 (200240) A61K031-00

EP 1216041 A2 20020626 (200249) EN A61K031-23

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI

CN 1390125 A 20030108 (200334) A61K031-23

MX 2002002728 A1 20020901 (200370) A61K031-00

EP 1216041 B1 20040204 (200410) EN A61K031-23

R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

DE 60008127 E 20040311 (200419) A61K031-23

ES 2213036 T3 20040816 (200455) A61K031-23

ADT WO 2001019356 A2 WO 2000-EP8731 20000907; EP 1090636 A1 EP  
1999-118173 19990913; AU 2000068430 A AU 2000-68430 20000907; BR  
2000013958 A BR 2000-13958 20000907, WO 2000-EP8731 20000907; EP  
1216041 A2 EP 2000-956522 20000907, WO 2000-EP8731 20000907; CN  
1390125 A CN 2000-815576 20000907; MX 2002002728 A1 WO 2000-EP8731  
20000907, MX 2002-2728 20020313; EP 1216041 B1 EP 2000-956522  
20000907, WO 2000-EP8731 20000907; DE 60008127 E DE  
2000-00008127 20000907, EP 2000-956522 20000907, WO 2000-EP8731  
20000907; ES 2213036 T3 EP 2000-956522 20000907

FDT AU 2000068430 A Based on WO 2001019356; BR 2000013958 A Based on WO  
2001019356; EP 1216041 A2 Based on WO 2001019356; MX 2002002728 A1 Based  
on WO 2001019356; EP 1216041 B1 Based on WO 2001019356; DE 60008127 E  
Based on EP 1216041, Based on WO 2001019356; ES 2213036 T3 Based on EP  
1216041

PRAI EP 1999-118173 19990913

IC ICM A61K031-00; A61K031-23

ICS A23L001-30; A61P003-00

AB WO 200119356 A UPAB: 20010528

NOVELTY - Composition for use as a medicament, food or nutritive product,  
comprises at least one **lipid**, which provides more than 35 %  
total energy of the composition.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for the  
preparation of a composition as above comprising blending the  
constituents, liquefying the blended mixture and homogenizing.

ACTIVITY - **Antiinflammatory**; antibacterial;  
immunosuppressive.

Tests were performed on an animal model of **sepsis** in rats  
to evaluate the body weight gain of rats post infection. Rats were fed  
either a 15 % (INF-15) **lipid** diet or a 35 % (INF-35)  
**lipid** diet for 6 days prior to infection and 10 days post  
infection. Two uninfected test groups were also fed a 15 % (PF-15) or 35 %  
(PF-35) **lipid** diets. Results are shown in the figure.

MECHANISM OF ACTION - None given.

USE - The composition is used in medicaments, foods or nutritive  
products for the treatment and prevention of **sepsis** or  
**inflammatory shock**. It may also be fed to animals e.g.  
pets.

ADVANTAGE - The composition can provide a high **lipid** diet  
to patients suffering from **inflammatory shock** or  
**sepsis**. The composition has a beneficial effect on recovery from  
acute **inflammatory** stress, and also on clinical parameters (body  
weight loss and nitrogen excretion).

DESCRIPTION OF DRAWING(S) - The figure shows the results of tests  
performed on an animal model of **sepsis** in rats to evaluate the  
body weight gain of rats fed a 15 or 35 % **lipid** diet, post  
infection.

Dwg.3/13

FS CPI

FA AB; GI; DCN

MC CPI: B04-B01B; B10-C04E; B14-C03; B14-S06;  
C04-B01B; C10-C04E; C14-C03; C14-S06; D03-G01;  
D03-H01T2

TECH UPTX: 20010528

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Composition: The  
**lipid** comprises 25-70 wt. % medium chain **triglycerides**



(MCT). The lipid comprises less than 15 wt. % saturated fatty acids excluding MCT. The composition comprises a n-6/n-3 fatty acid ratio of 2/1 to 7/1. The composition comprises at least one n-3 fatty acid selected from alpha-linolenic acid, eicosapentaenoic acid (EDA), docosapentaenoic acid (DPA) and docosahexaenoic acid (DHA). The composition comprises at least one n-6 fatty acid selected from linoleic acid (18:2), gamma-linolenic acid (18:3), dihomo-gamma-linolenic acid (18:4), or arachidonic acid. The composition may be administered enterally and further comprise a carrier, diluent or adjuvant.

ABEX UPTX: 20010528

ADMINISTRATION - Administration is enteral (claimed).

EXAMPLE - A typical composition was prepared comprising 18 % protein, 37 % carbohydrate, and 45 % lipid. The lipid part of the composition contained (g/L): saturated lipid (including medium chain triglycerides) (42.8); monounsaturated lipid (23.3); polyunsaturated lipid (9.0); linoleic acid (18:2; n-6) (6.8); and alpha-linolenic acid (18:3; n-3) (2.3). The caloric density of the composition was 1.5 Kcal/ml.

L142 ANSWER 3 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1999-551195 [46] WPIX

DNC C1999-160839

TI Use of omega 3 fatty acid for treating disorders in which docosahexaenoic acid levels are affected.

DC B05

IN ALVAREZ, J G; FREEDMAN, S

PA (BETH-N) BETH ISRAEL DEACONESS MEDICAL CENT

CYC 82

PI WO 9945916 A2 19990916 (199946)\* EN 50 A61K031-20 <--  
 RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL  
 OA PT SD SE SL SZ UG ZW  
 W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD  
 GE GH GM HR HU ID IL IN IS JP KE KG KP LK LR LS LT LU LV MD MG MK  
 MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US  
 UZ VN YU ZW  
 AU 9929824 A 19990927 (200006) A61K031-20 <--  
 BR 9908697 A 20001121 (200065) A61K031-20  
 EP 1061911 A2 20001227 (200102) EN A61K031-20  
 R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
 RO SE SI  
 US 6180671 B1 20010130 (200108) A61K031-20  
 JP 2002506026 W 20020226 (200219) 67 A61K031-202  
 MX 2000008475 A1 20011101 (200279) A23L001-30 <--  
 US 6552081 B1 20030422 (200330) A61K031-20  
 AU 762660 B 20030703 (200354) A61K031-20  
 US 2004127567 A1 20040701 (200444) A61K031-202

ADT WO 9945916 A2 WO 1999-US4722 19990303; AU 9929824 A AU 1999-29824 19990303; BR 9908697 A BR 1999-8697 19990303, WO 1999-US4722 19990303; EP 1061911 A2 EP 1999-911098 19990303, WO 1999-US4722 19990303; US 6180671 B1 CIP of US 1998-37222 19980310, CIP of US 1999-231479 19990114, US 1999-248471 19990211; JP 2002506026 W WO 1999-US4722 19990303, JP 2000-535331 19990303; MX 2000008475 A1 MX 2000-8475 20000830; US 6552081 B1 CIP of US 1998-37222 19980310, CIP of US 1999-231479 19990114, Div ex US 1999-248471 19990211, US 2000-706404 20001103; AU 762660 B AU 1999-29824 19990303; US 2004127567 A1 CIP of US 1998-37222 19980310,

CIP of US 1999-231479 19990114, Div ex US 1999-248471

19990211, Div ex US 2000-706404 20001103, US 2003-410511 20030408

FDT AU 9929824 A Based on WO 9945916; BR 9908697 A Based on WO 9945916; EP 1061911 A2 Based on WO 9945916; JP 2002506026 W Based on WO 9945916; US 6552081 B1 Div ex US 6180671; AU 762660 B Previous Publ. AU 9929824, Based on WO 9945916; US 2004127567 A1 Div ex US 6180671, Div ex US 6552081

PRAI US 1999-248471 19990211; US 1998-37222

19980310; US 1999-231479 19990114; US

2000-706404 20001103; US 2003-410511 20030408

IC ICM A23L001-30; A61K031-20; A61K031-202

ICS A61K031-23; A61P001-00; A61P001-04; A61P011-06; A61P029-00; A61P043-00

AB WO 9945916 A UPAB: 19991110

NOVELTY - Treatment of disorders in which **docosahexaenoic acid** (DHA) levels are affected comprises administering an **omega 3 fatty acid** having 22-24 C atoms and 5 or more double bonds.

USE - The **fatty acid** is administered to treat disorders in which DHA levels are affected e.g. defects in the CF or CFTR gene, cystic fibrosis, a chronic **inflammatory** disorder e.g. ulcerative colitis, Crohn's disease, chronic pancreatitis, asthma, rheumatoid arthritis, chronic gastritis, lowered fetal surfactant levels, respiratory distress syndrome, hypertrophy of the small intestine or to restore normal morphology to a cell or tissue which exhibits a disease morphology associated with disorder in which DHA levels are affected. The method is especially useful for ameliorating the effects of cystic fibrosis in a newborn.

Dwg.5/8

FS CPI

FA AB; GI; DCN

MC CPI: B10-C04E; B14-C03; B14-C06; B14-C09; B14-E08; B14-E12; B14-G02A; B14-G02D; B14-K01; B14-K01D

TECH UPTX: 19991110

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Compounds: The

**omega 3 fatty acid** is DHA or a DHA

precursor e.g. **docosapentaenoic acid**, tetracosapentaenoic acid and tetracosahexaenoic acid. The acid may be in a form easily incorporated into the tissue such as a derivative or a structured **lipid**. Preferably the structured **lipid** has the DHA located at the R2 position of the glycerol backbone.

ABEX UPTX: 19991110

ADMINISTRATION - Administration is preferably oral. DHA levels are raised to at least about 170 (preferably 200-500) g/ml. The DHA is administered at about 0.3-5% of the total calorific intake and should produce a pancreatic **arachidonic acid** (AA)/DHA ratio of about 0.2-1.5 or a lung AA/DHA ratio of 0.1-1.6 or a blood AA/DHA ratio of 0.1-1.6.

EXAMPLE - The figures show the effects of oral DHA administration at various concentrations on **arachidonic acid** (AA) and DHA levels in preparations of pancreatic acini and lung cells from CFTR (-/-) mice (CF) compared with wild type mice (WT).

L142 ANSWER 4 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1999-372252 [32] WPIX

DNC C1999-110070

TI Fat mixture with polyunsaturated fats and **oils**, useful for treating e.g. **inflammatory** diseases.

DC B05 D13

IN BOEHN, G; FARWER, S; KLIEM, M; KOHN, G; SAWATZKI, G; BOEHM, G

PA (NUTR-N) NUTRICIA NV

CYC 34

PI	DE 19757414	A1 19990701 (199932)*	10	A23L001-29	<--
	WO 9933355	A2 19990708 (199934)	GE	A23L001-00	<--

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
W: AL AU BR CA CN IN JP LT LV MK NO NZ RO SI US

AU 9924162	A	19990719 (199951)	A23L001-00	<--
NO 2000003265	A	20000622 (200050)	A23D000-00	
EP 1041896	A2	20001011 (200052) GE	A23L001-30	<--
R: AT BE CH DE DK ES FR GB IE IT LI NL SE				
BR 9814467	A	20001010 (200055)	A23L001-00	<--
CN 1282223	A	20010131 (200131)	A23L001-30	<--
JP 2001526908	W	20011225 (200204) 23	A23D009-007	
EP 1041896	B1	20020220 (200214) GE	A23L001-30	<--
R: AT BE CH DE DK ES FR GB IE IT LI NL SE				
DE 59803153	G	20020328 (200229)	A23L001-30	<--
ES 2173673	T3	20021016 (200279)	A23L001-30	<--
NZ 505608	A	20021220 (200309)	A23L001-00	<--
AU 758079	B	20030313 (200328)	A23L001-00	<--
NO 316302	B1	20040112 (200406)	A23D009-00	

ADT DE 19757414 A1 DE 1997-1057414 19971223; WO 9933355 A2 WO 1998-EP8409 19981222; AU 9924162 A AU 1999-24162 19981222; NO 2000003265 A WO 1998-EP8409 19981222, NO 2000-3265 20000622; EP 1041896 A2 EP 1998-966662 19981222, WO 1998-EP8409 19981222; BR 9814467 A BR 1998-14467 19981222, WO 1998-EP8409 19981222; CN 1282223 A CN 1998-812474 19981222; JP 2001526908 W WO 1998-EP8409 19981222, JP 2000-526127 19981222; EP 1041896 B1 EP 1998-966662 19981222, WO 1998-EP8409 19981222; DE 59803153 G DE 1998-503153 19981222, EP 1998-966662 19981222, WO 1998-EP8409 19981222; ES 2173673 T3 EP 1998-966662 19981222; NZ 505608 A NZ 1998-505608 19981222, WO 1998-EP8409 19981222; AU 758079 B AU 1999-24162 19981222; NO 316302 B1 WO 1998-EP8409 19981222, NO 2000-3265 20000622

FDT AU 9924162 A Based on WO 9933355; EP 1041896 A2 Based on WO 9933355; BR 9814467 A Based on WO 9933355; JP 2001526908 W Based on WO 9933355; EP 1041896 B1 Based on WO 9933355; DE 59803153 G Based on EP 1041896, Based on WO 9933355; ES 2173673 T3 Based on EP 1041896; NZ 505608 A Based on WO 9933355; AU 758079 B Previous Publ. AU 9924162, Based on WO 9933355; NO 316302 B1 Previous Publ. NO 2000003265

PRAI DE 1997-19757414 19971223

IC ICM A23D000-00; A23D009-00; A23D009-007; A23L001-00; A23L001-29; A23L001-30

ICS A61K031-20; A61K045-00; A61P003-06; A61P029-00; A61P037-04

AB DE 19757414 A UPAB: 19990813

NOVELTY - A fat mixture (I) comprising **oils**, fats and/or lecithins with polyunsaturated fatty acids which comprise **gamma-linolenic acid** (20-50 weight%), stearidonic acid (15-50 weight%) and **eicosapentaenoic acid** (20-50 weight%) at 10-500 mg/g.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for dietary foods containing (I).

USE - (I) is used in the dietary treatment of patients with chronic **inflammatory** diseases, disturbances of **lipid** metabolism, weak immune system function and/or limited lipolytical capacity of the gastrointestinal tract (claimed).

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B04-B01B; B10-C04E; B14-C03; B14-E10; B14-G01; D03-C01; D03-H01T2

TECH UPTX: 19990813

TECHNOLOGY FOCUS - FOOD - Preferred Materials: (I) comprises **gamma-linolenic acid** and **eicosapentaenoic acid** (35-45 wt.%) and stearidonic acid (15-25 wt.% of the total of three acids). (I) comprises **phospholipids** (1-10 wt.% of the total **lipids**). The dietary foods are in the form of fatty emulsions, ready made meals, liquid food, reconstituted or reconstitutuable powder, especially for

enteral or oral application, as bar or spread. The dietary food comprises an energy supply of 0.5-3 kcal/ml.

L142 ANSWER 5 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1998-178507 [16] WPIX

DNC C1998-057272

TI Enteral nutrient compositions for optimised absorption and wound healing in e.g. trauma patients - comprising partially hydrolysed proteins, free amino acids, carbohydrates and **lipids** including **omega-3 fatty acids**.

DC B05 D13

IN ALEXANDER, J; GRAY, D; MARK, D A; SCHMELKIN, N S; TWYMAN, D

PA (NEST) NESTEC LTD

CYC 1

PI US 5723446 A 19980303 (199816)\* 5 A61K038-00 <--

ADT US 5723446 A Cont of US 1993-172857 19931223, US

1996-680703 19960717

PRAI US 1993-172857 19931223; US 1996-680703

19960717

IC ICM A61K038-00

ICS A23G003-00; A23J001-00

AB US 5723446 A UPAB: 19980421

Providing enteral nutrition to trauma, burn or post-surgery patients comprises enterally administering a composition containing: (a) a protein source comprising 22-28(25)% of the total calories; (b) a carbohydrate source comprising 35-40% of the total calories; and (c) a **lipid** source comprising 33-45% of the total calories including a source of medium chain **triglycerides** comprising 40-60% of the

**lipid** source, a source of **omega -3**

**fatty acids** providing 2.2-3% of the total calories, and

a source of **omega -6 fatty acids**.

The composition provides sources of arginine, proline, beta -carotene and/or cysteine. The protein source includes a majority(68-88%) of protein calories as partially hydrolysed proteins and does not contain whole proteins. The composition is fed to a patient through a tube and has a caloric density of 1.3-1.5 kilocalories/ml.

ADVANTAGE - The composition has high protein and **lipid** content and high caloric density to meet energy needs, and has reduced water and carbohydrate content, reducing the risk of diarrhea. Nutrient malabsorption is reduced by the absence of whole proteins and by the use of protein hydrosylate, free amino acids and medium chain **triglycerides**. The ready to use formulation does not require mixing, thus reducing the risk of bacterial contamination. The composition minimises **inflammatory** reactions.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B03-A; **B04-B01B**; B04-D01; B04-N02; B05-B01P; B07-D03; B10-A17; B10-B02D; B10-C04E; B10-G02; B14-E11; B14-N17; D03-F06; **D03-H01T2**

L142 ANSWER 6 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1997-299573 [28] WPIX

DNC C1997-097229

TI Isotonic **lipid** emulsion for parenteral application for nourishment, - in patients with, e.g. exacerbated **inflammatory** reaction or increased risk of vascular thrombosis, based on plant and **fish oil** and middle-chain tri **glyceride** derivatives.

DC B04 D13

IN CARPENTIER, Y A; JUNGINGER, M; NEHNE, J; PSCHERER, G; JUNINGER, M

PA (BINT) BRAUN MELSUNGEN AG B

CYC 66

PI DE 19648566 A1 19970605 (199728)\* 13 A61K035-78 <--  
 WO 9719683 A1 19970605 (199728) EN 32 A61K031-19 <--  
 RW: AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD  
 SE SZ UG  
 W: AL AU BA BB BG BR CA CN CU CZ EE GE HU IL IS JP KP KR LC LK LR LT  
 LV MG MK MN MX NO NZ PL RO SG SI SK TR TT UA US UZ VN  
 AU 9676972 A 19970619 (199741) A61K031-19 <--  
 EP 863754 A1 19980916 (199841) EN A61K031-19 <--  
 R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE SI  
 CZ 9801618 A3 19981111 (199851) A61K031-19 <--  
 AU 701736 B 19990204 (199917) A61K031-19 <--  
 CN 1202823 A 19981223 (199919) A61K031-19 <--  
 NZ 322927 A 19990629 (199931) A23L001-30 <--  
 BR 9611826 A 19990713 (199939) A61K031-19 <--  
 US 6008248 A 19991228 (200007) A61K031-19 <--  
 JP 2000500769 W 20000125 (200016) 28 A61K031-201  
 HU 9903426 A2 20000228 (200020) A61K031-19  
 MX 9804127 A1 19981201 (200024) A61K031-19 <--  
 CA 2236422 C 20021119 (200304) EN A61K031-19  
 EP 863754 B1 20030102 (200310) EN A61K031-19  
 R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE SI  
 DE 69625616 E 20030206 (200318) A61K031-19  
 ES 2189891 T3 20030716 (200356) A61K031-19  
 MX 213257 B 20030317 (200413) A23L001-30 <--

ADT DE 19648566 A1 DE 1996-1048566 19961123; WO 9719683 A1 WO  
 1996-EP5184 19961123; AU 9676972 A AU 1996-76972 19961123;  
 EP 863754 A1 EP 1996-939912 19961123, WO 1996-EP5184  
 19961123; CZ 9801618 A3 WO 1996-EP5184 19961123, CZ  
 1998-1618 19961123; AU 701736 B AU 1996-76972 19961123; CN  
 1202823 A CN 1996-198603 19961123; NZ 322927 A NZ  
 1996-322927 19961123, WO 1996-EP5184 19961123; BR 9611826 A  
 BR 1996-11826 19961123, WO 1996-EP5184 19961123; US  
 6008248 A WO 1996-EP5184 19961123, US 1998-43166  
 19980312; JP 2000500769 W WO 1996-EP5184 19961123, JP  
 1997-520149 19961123; HU 9903426 A2 WO 1996-EP5184 19961123  
 , HU 1999-3426 19961123; MX 9804127 A1 MX 1998-4127  
 19980525; CA 2236422 C CA 1996-2236422 19961123, WO  
 1996-EP5184 19961123; EP 863754 B1 EP 1996-939912 19961123,  
 WO 1996-EP5184 19961123; DE 69625616 E DE 1996-625616  
 19961123, EP 1996-939912 19961123, WO 1996-EP5184  
 19961123; ES 2189891 T3 EP 1996-939912 19961123; MX 213257  
 B WO 1996-EP5184 19961123, MX 1998-4127 19980525

FDT AU 9676972 A Based on WO 9719683; EP 863754 A1 Based on WO 9719683; CZ  
 9801618 A3 Based on WO 9719683; AU 701736 B Previous Publ. AU 9676972,  
 Based on WO 9719683; NZ 322927 A Based on WO 9719683; BR 9611826 A Based  
 on WO 9719683; US 6008248 A Based on WO 9719683; JP 2000500769 W Based on  
 WO 9719683; HU 9903426 A2 Based on WO 9719683; CA 2236422 C Based on WO  
 9719683; EP 863754 B1 Based on WO 9719683; DE 69625616 E Based on EP  
 863754, Based on WO 9719683; ES 2189891 T3 Based on EP 863754

PRAI DE 1995-19544310 19951128

REP DE 3409793; DE 3721137; EP 311091; EP 687418; FR 2542613; US 5444054; US  
 5470839

IC ICM A61K031-19; A61K031-201; A61K035-78  
 ICS A61K009-107; A61K031-23; A61K031-231; A61K035-60; A61K045-00;  
 A61P003-02; A61P005-30; A61P007-02; A61P009-00; A61P029-00

ICA A23D007-00; A23L001-30

AB DE 19648566 A UPAB: 19970709

Isotonic lipid emulsion for parenteral application consisting of  
 middle chain triglycerides (30-60 weight%) and at least 1 plant  
 oil containing omega-6-fatty  
 acids (35-65 weight%) and a fish oil containing  
 omega-3-fatty acids (5-20 weight%).

USE - The composition is used for parenteral nourishment in patients

with exacerbated **inflammatory** reaction, increased risk of vascular thrombosis, heart arrhythmia, post operative or post traumatic conditions or **inflammatory** disease (all claimed).

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B04-B01C1; B04-B01C2; B12-M03; B14-C03; B14-F01A; B14-F04;  
**D03-H01T2**

L142 ANSWER 7 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1997-021672 [02] WPIX

CR 1996-506074 [50]; 1996-506079 [50]; 1996-506082 [50]; 1997-021670 [49];  
1997-021671 [49]

DNC C1997-007027

TI gem-Diol ester(s) of polyunsaturated fatty acids e.g. **gamma linolenic** acid - for pharmaceutical, food and cosmetic use.

DC B04 B05 C03 D13 D21

IN HORROBIN, D F; KNOWLES, P; MANKU, M; MCMORDIE, A; PITT, A; REDDEN, P

PA (SCOT-N) SCOTIA HOLDINGS PLC

CYC 1

PI ZA 9603433 A 19961030 (199702)\* EN 41 A23L000-00 <--

ADT ZA 9603433 A ZA 1996-3433 19960430

PRAI **GB 1995-8823 19950501**

IC ICM A23L000-00

ICS A61K000-00; C07C000-00

AB ZA 9603433 A UPAB: 19970212

gem-Diol esters of formula R10-CHR3-OR2 (I) are new. R1 = acyl gp. derived from a 16-30C **fatty acid** containing two or more cis or trans double bonds, partic. an **n-6** or **n-3** series essential **fatty acid** or conjugated **linoleic** acid (cLA) or columbinic acid (CA) or parinaric acid; R2 = R1 or any other nutrient, drug or bioactive residue; R3 = H or hydrocarbyl opt. containing heteroatoms, pref. alkyl, partic. 1-4C alkyl.

USE - (I) where R1 = acyl derived from **gamma**

**linolenic** acid (GLA) or **dihomo gamma**

**linolenic** acid (DGLA) and R2 = acyl derived from GLA, DGLA,

stearidonic acid (SA), **eicosapentaenoic** acid (EPA),

**docosahexaenoic** acid (DHA), cLA or CA are useful as food

components, nutritional supplements, food additives, components of

clinical nutrition prods. for enteral or parenteral admin. and cosmetic

components, especially for treating (a) complications of diabetes, especially

neuropathy and retinopathy; and improvement of responses to insulin in

diabetes and pre-diabetes; (b) cancers; (c) osteoarthritis; (d) rheumatoid

arthritis; (e) other **inflammatory** and auto-immune diseases e.g.

Sjogren's syndrome, systemic lupus, ulcerative colitis, Crohn's disease

and uveitis; (f) respiratory diseases e.g. asthma; (g) neurological

disorders e.g. multiple sclerosis, Parkinson's disease and Huntington's

chorea; (h) renal and urinary tract disorders; (i) cardiovascular

disorders; (j) degenerative diseases of the eye e.g. retinitis pigmentosa

and senile macular degeneration; (k) psychiatric disorders including

schizophrenia, Alzheimer's disease, attention deficit disorder, alcoholism

and depression; (l) prostatic hypertrophy and prostatitis; (m) impotence

and male infertility; (n) mastalgia; (o) male pattern baldness; (p)

osteoporosis; (q) dermatological and allergic disorders; (r) dyslexia and

other learning disabilities; and (s) cancer cachexia. (I) where R1 = acyl

derived from GLA, DGLA, **arachidonic** acid (AA), SA, cLA, EPA or

DHA and R2 = one of the following agents are useful for treating any

disease especially the following disorders, and other uses mentioned: (a)

tryptophan for psychiatric, neurological, behavioural, pain and other

disorders and especially depression, sleep and migraine; (b) phenylalanine for

depression, multiple sclerosis and chronic fatigue syndrome; (c) arginine

for diseases in which the production of nitric oxide is defective; (d)

carnitine or carnitine derivs. for muscle weakness, cardiac failure,

chronic fatigue syndrome, Alzheimer's disease, and peripheral neuropathies; (e) any other amino acid or related substance or aminolevulinic acid or derivative thereof for cancers; (f) adenylosuccinate or related substances for muscular dystrophy, cardiac failure, chronic fatigue and Alzheimer's disease and other dementias; (g) aspirin, salicylic acid, indomethacin, ibuprofen, or any other non-steroidal anti-inflammatory drug for inflammatory disorders or pain, of Alzheimer's disease and other dementias and of any disease in which platelet aggregation should be inhibited; (h) any antibiotic for the treatment of any appropriate infectious disease but especially tetracycline, clindamycin, minocycline, chlortetracycline and erythromycin for the treatment of acne; (i) any antimalarial or anti-protozoal drug especially chloroquine, mepacrine, quinacrine and mefloquine for the treatment of malaria, protozoal disorders, inflammatory disorders and schizophrenia; (j) any antifungal drug especially metronidazole and antifungal imidazoles and nitroimidazoles and amphotericin for the treatment of fungal infections of various types; (k) any anti-inflammatory steroid especially hydrocortisone and betamethasone for the treatment of skin disorders and beclomethasone and budesonide for the treatment of asthma; (l) any gonadal steroid especially oestrogens and progestogens for the

treatment

of ovarian deficiency and osteoporosis and androgens for the treatment of testicular deficiency; (m) any adrenal steroid especially

dehydroepiandrosterone

for the treatment of disorders associated with ageing; (n) any retinoid especially tretinoin and isotretinoin for the treatment of dermatological disorders and for use in skin care; (o) any anticancer agent for the treatment of cancer; (p) any antipsychotic agent for the treatment of schizophrenia and other psychoses; (q) any antidepressive agent for the treatment of depression; (r) any anti-anxiety agent especially for the

treatment

of anxiety and panic attacks; (s) any immunosuppressive agent especially cyclosporine and tacrolimus for the control of immunity after organ transplantation and for the treatment of autoimmune and inflammatory disorders including psoriasis, eczema, asthma, rheumatoid arthritis and inflammatory bowel disease; (t) any proton pump inhibitor or H2 antagonist especially diseases associated with excess gastric acid production or reduced defences against gastric acidity; (u) any diuretic to treat fluid retention and hypertension; (v) any calcium antagonist or angiotensin converting enzyme inhibitor or beta blocker to treat cardiovascular disease; (w) antiepileptic drug especially phenytoin, carbamazepine or lamotrigine to treat epilepsy; (x) any hypolipidaemic agent especially fibrates and statins for cholesterol lowering; (y) any oral hypoglycaemic for diabetes management; (z) any bisphosphonates for management of osteoporosis or Paget's disease; (aa) any contrast agents for radiology; (bb) any peptide or protein for treatment using these diseases.

ADVANTAGE - Transport through lipid membranes, e.g. of cells, of the skin or the blood-brain barrier, is enhanced.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B10-G02; C10-G02; B14-C03; C14-C03; B14-C09A; C14-C09A; B14-C09B; C14-C09B; B14-E10C; C14-E10C; B14-E11; C14-E11; B14-G02D; C14-G02D; B14-H01; C14-H01; B14-J01A; C14-J01A; B14-K01A; C14-K01A; B14-N03; C14-N03; B14-N07A; C14-N07A; B14-S04; C14-S04; D03-H01T2; D08-B09A

L142 ANSWER 8 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1996-031549 [04] WPIX

DNC C1996-010844

TI Lipid compsn. for limiting injury response in trauma, burns etc.  
- comprises medium chain tri glyceride and omega-

6 and -3 fatty acid sources, useful for enteral and parenteral admin..

DC B05 D13  
IN TRIMBO, S; TRIMBO, S L  
PA (CLIN-N) CLINTEC NUTRITION CO; (NEST) SOCIETE DES PRODUITS NESTLE SA;  
(NEST) NESTEC LTD

CYC 18

PI EP 687418 A2 19951220 (199604)\* EN 5 A23D009-00 <--  
R: AT BE CH DE DK ES FR GB GR IE IT LI LU NL PT SE  
CA 2147302 A 19951022 (199610) A61K031-22 <--  
US 5574065 A 19961112 (199651) 5 A61K031-22 <--  
EP 687418 A3 19970514 (199731) A23D009-00 <--  
US 5700837 A 19971223 (199806) 6 A61K031-22 <--

ADT EP 687418 A2 EP 1995-200987 19950418; CA 2147302 A CA  
1995-2147302 19950419; US 5574065 A US 1994-230592 19940421  
; EP 687418 A3 EP 1995-200987 19950418; US 5700837 A Cont  
of US 1994-230592 19940421, US 1996-612980 19960305

FDT US 5700837 A Cont of US 5574065

PRAI US 1994-230592 19940421; US 1996-612980  
19960305

REP DE 3721137; EP 311091; EP 484266; US 5444054; US 5470839

IC ICM A23D009-00; A61K031-22  
ICS A23L001-30; A61K031-00; A61K031-19; A61K031-20; A61K031-225

AB EP 687418 A UPAB: 19970522  
A lipid compsn. comprises medium chain triglyceride,  
an omega -6 fatty acid source and  
an omega -3 fatty acid source.

USE - The compsns limit the injury response in patients suffering  
from trauma, burns and/or sepsis. They may be part of a complete  
diet and may be in the form of a parenteral emulsion or an enteral compsn.  
Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B04-B01C1; B04-B01C2; B10-G02; B14-N17A; B14-N17B; B14-S06;  
D03-H01T2

ABEQ US 5574065 A UPAB: 19961219

Lipid compsn. comprises:

medium chain triglycerides, the medium chain  
triglycerides comprise approx. 25 wt.% of the compsn.;  
an omega-3 fatty acid source,  
the omega-3 fatty acid source  
comprises approx. 35 wt.% of the compsn., and  
an omega-6 fatty acid source,  
the omega-6 fatty acid source  
comprises approx. 40 wt.% of the compsn.  
Dwg.0/0

ABEQ US 5700837 A UPAB: 19980209

A lipid compsn. comprises medium chain triglyceride,  
an omega -6 fatty acid source and  
an omega -3 fatty acid source.

USE - The compsns limit the injury response in patients suffering  
from trauma, burns and/or sepsis. They may be part of a complete  
diet and may be in the form of a parenteral emulsion or an enteral compsn.  
Dwg.0/0

L142 ANSWER 9 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1995-310360 [40] WPIX

CR 1995-358387 [46]; 1999-589273 [50]; 2003-127890 [12]

DNC C1995-138367

TI Treatment of ulcerative colitis and/or colon inflammation -  
using nutritional prod. containing blend of n-6 and  
n-3 fatty acids with carbohydrate  
metabolised to short chain fatty acids in colon.



DC B05 D13  
 IN DEMICHELE, S J; GARLEB, K A; FULLER, M K; MCEWEN, J W  
 PA (ABBO) ABBOTT LAB  
 CYC 20  
 PI US 5444054 A 19950822 (199540)\* 23 A61K031-70 <--  
 EP 754001 A1 19970122 (199709) EN A23L001-30 <--  
 R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE  
 BR 1101101 A3 19980512 (199828) A61K031-20 <--  
 MX 9605144 A1 19971201 (199936) A23L001-30 <--  
 MX 192837 B 19990730 (200061) A61K031-715 <--  
 ES 2171535 T3 20020916 (200270) A23L001-30 <--  
 ADT US 5444054 A US 1994-221440 19940401; EP 754001 A1 EP  
 1995-911135 19950306; WO 1995-US2657 19950306; BR 1101101  
 A3 BR 1997-1101101 19970514; MX 9605144 A1 MX 1996-5144  
 19961025; MX 192837 B MX 1996-5144 19950306; ES 2171535 T3  
 EP 1995-911135 19950306  
 FDT EP 754001 A1 Based on WO 9526646; ES 2171535 T3 Based on EP 754001  
 PRAI US 1994-221440 19940401; US 1994-221349  
 19940401; WO 1995-US2657 19950306  
 REP 2.Jnl.Ref; EP 115419; EP 404058; JP 4066528; US 5104677; WO 9012080; WO  
 9321912  
 IC ICM A23L001-30; A61K031-20; A61K031-70; A61K031-715  
 ICS A23G003-000; A23L001-305; A23L001-308  
 AB US 5444054 A UPAB: 20030218  
 Method of improving the nutritional status and reversing the  
 characteristic diarrhoea and **inflammatory** condition in a mammal  
 having ulcerative colitis or **inflammation** of the colon is  
 claimed. The method comprises feeding the patient enterally with a liquid  
 nutritional prod. (A1) or (A2). (A1) comprises (a) an **oil** blend  
 (a1) or (a'1) and a source of indigestible carbohydrate which is  
 metabolised to short chain **fatty acids** (SCFA) by  
 microorganisms present in the human colon (b1) and which contains dietary  
 fibre(s) and/or indigestible oligosaccharides. (A2) comprises an  
**oil** blend (a2), indigestible carbohydrate (b2) as above which is  
 gum arabic, soy polysaccharide, hydrolysed inulin, fructooligosaccharide  
 (FOS) or xylooligosaccharide (XOS). ; (c) nutrient(s) selected from beta  
 -carotene, vitamin E, vitamin C, taurine and selenium; and (d) protein.  
 (a1) contains at least 25 weight % of **oils** containing eicosapentenoic  
 acid (20:5n3) (EPA) and **docosahexaenoic** acid (22:6n3) (DXE) and  
 (A1) also contains (c). (a'1) comprises an **oil** blend containing the  
 individual **fatty acids** oleic acid (18:1n9)  
 11.5-15.7%; **linoleic** acid (18:2n6) 6.6-9.0%; **alpha -**  
**linolenic** acid (18:3n3) 1.5-2.1%; EPA 15.1-20.5%; and DXE  
 6.3-8.6%. (a2) comprises 5-40% canola **oil**, 10-50% medium chain  
**triglycerides**, 25-80% **fish oil** 3-30% soybean  
**oil**, and 2-6% soy lecithin.  
 ADVANTAGE - The prod. is free from the side effects of drug therapy  
 as used at present, using **antiinflammatory**, antibiotic, and/or  
 immunosuppressive agents; and of total parenteral nutrition. The SCFA are  
 beneficial; the carbohydrate (b) is metabolised to provide these without  
 the need for rectal admin. The condition is also through to stem from  
**arachidonic** acid-derived **inflammatory** mediators; there  
 is evidence that n-3 acids inhibit this pathway to  
 give an **antiinflammatory** effect, and also that the SCFA improve  
 incorporation of n-3 acids into colonocytes, reducing  
 mucosal irritation. The components (c) are either antioxidants or are  
 required for functioning of antioxidant enzymes to reduce oxygen radical  
 mediated damage.  
 Dwg.0/5  
 FS CPI  
 FA AB; DCN  
 MC CPI: B03-A; B03-F; B03-H; **B04-B01B**; B04-B01C1; B04-B01C2;  
 B04-C02D; B04-C02X; B04-N02; B05-A03B; B10-C02; B10-C04E; B14-C03;

B14-E08; B14-E10C; B14-E11; B14-S08

L142 ANSWER 10 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN  
 AN 1995-122870 [16] WPIX  
 CR 1997-502374 [46]; 1998-347237 [30]  
 DNC C1995-056070  
 TI Enteral compsns. containing poly unsatd. fat and saponin - for treating or preventing infection or **inflammation**.  
 DC B05  
 IN CHAVALI, S; FORSE, R A; FORSE, R  
 PA (NEWE-N) NEW ENGLAND DEACONESS HOSPITAL  
 CYC 63  
 PI US 5397778 A 19950314 (199516)\* 6 A61K031-38 <--  
 WO 9522971 A1 19950831 (199540) EN 15 A61K031-36 <--  
 RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG  
 W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG  
 KP KR KZ LK LR LT LU LV MD MG MN MW MX NL NO NZ PL PT RO RU SD SE  
 SI SK TJ TT UA UZ VN  
 WO 9528163 A1 19951026 (199548) EN 17 A61K031-70 <--  
 RW: AT BE CH DE DK ES FR GB GR IE IT KE LU MC MW NL OA PT SD SE SZ UG  
 W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG  
 KP KR KZ LK LR LT LU LV MD MG MN MW MX NL NO NZ PL PT RO RU SD SE  
 SG SI SK TJ TT UA UZ VN  
 AU 9519282 A 19950911 (199550) A61K031-36 <--  
 AU 9522744 A 19951110 (199607) A61K031-70 <--  
 EP 804207 A1 19971105 (199749) EN A61K031-70 <--  
 R: BE CH DE FR GB IT LI NL  
 KR 97701043 A 19970317 (199813) A61K031-36 <--  
 JP 10500937 W 19980127 (199814) 17 A61K031-34 <--  
 JP 10504016 W 19980414 (199825) 23 A61K031-70 <--  
 EP 873116 A1 19981028 (199847) EN A61K031-36 <--  
 R: BE CH DE FR GB IT LI NL  
 ADT US 5397778 A CIP of US 1994-201682 19940225, US 1994-228599  
 19940415; WO 9522971 A1 WO 1995-US2246 19950222; WO 9528163  
 A1 WO 1995-US3894 19950329; AU 9519282 A AU 1995-19282  
 19950222; AU 9522744 A AU 1995-22744 19950329; EP 804207 A1  
 EP 1995-916134 19950329, WO 1995-US3894 19950329; KR  
 97701043 A WO 1995-US2246 19950222, KR 1996-704747  
 19960826; JP 10500937 W JP 1995-522448 19950222, WO  
 1995-US2246 19950222; JP 10504016 W JP 1995-526982 19950329  
 , WO 1995-US3894 19950329; EP 873116 A1 EP 1995-911881  
 19950222, WO 1995-US2246 19950222  
 FDT AU 9519282 A Based on WO 9522971; AU 9522744 A Based on WO 9528163; EP  
 804207 A1 Based on WO 9528163; KR 97701043 A Based on WO 9522971; JP  
 10500937 W Based on WO 9522971; JP 10504016 W Based on WO 9528163; EP  
 873116 A1 Based on WO 9522971  
 PRAI US 1994-228599 19940415; US 1994-201682  
 19940225  
 REP 12Jnl.Ref; DE 4017766; EP 387000; JP 2273622; JP 63157934; US 4752618; WO  
 8902275; DE 3001435; EP 183674; EP 253198; EP 305243; EP 534178; GB  
 2041942; WO 9523167  
 IC ICM A61K031-34; A61K031-36; A61K031-38; A61K031-70  
 ICS A23L001-03; A23L001-30; A23L001-302;  
 A23L001-304; A61K031-23; A61K033-00; A61K035-60; A61K035-78  
 ICA C07D493-04  
 AB US 5397778 A UPAB: 19980730  
 Method for treating and preventing infection in persons at risk comprises  
 enteral admin. of a formulation containing Quil A saponin (I) as its active  
 ingredient, in conjunction with a source of dietary polyunsatd. fatty  
 acids (II), provided that (II) constitute at least a significant part of  
 the fat fed to the persons. Also claimed is an enteral formulation for  
 treatment of infection or **inflammation** in a patient, having (I)  
 as an active ingredient and comprising a source of (II), with the above

proviso.

The source of (II) is pref. a **fish oil** or vegetable oil rich in omega-3 fatty acids, especially a **fish oil** containing at least 10% omega-3 fatty acids. A lignan selected from sesamin, episesamin, sesaminol, episesaminol and sesamol is pref. administered in conjunction with the enteral formulation

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: B04-A07E; **B04-B01B**; B10-C04E; B14-A01; B14-A02; B14-C03; B14-N

L142 ANSWER 11 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1992-433346 [52] WPIX

DNC C1992-192348

TI New emulsion for reducing blood tri **glyceride** and cholesterol levels - comprises vegetable and/or marine oil, an aqueous phase and a **phospholipid**.

DC B05 D13 E11 E17

IN LARSSON-BACKSTROEM, C; LARSSON-BACKSTROM, C; LARSSONBACKSTROEM, C

PA (KABI) KABI PHARMACIA AB; (FREP) FRESSENIUS KABI AB; (PHAA) PHARMACIA & UPJOHN AB; (PHAA) PHARMACIA AB

CYC 25

PI WO 9221335 A1 19921210 (199252)\* EN 32 A61K031-19 <--

RW: AT BE CH DE DK ES FR GB GR IT LU MC NL SE

W: AU CA FI JP KR NO US

AU 9219101 A 19930108 (199315) A61K031-19 <--

PT 100524 A 19930831 (199338) B01F003-08 <--

NZ 242697 A 19940225 (199411) C08L091-00 <--

JP 06508123 W 19940914 (199441) A61K031-20 <--

AU 657969 B 19950330 (199521) A23D007-02 <--

EP 660708 A1 19950705 (199531) EN A61K031-19 <--

R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE

US 5434183 A 19950718 (199534) 10 A61K031-66 <--

SG 64864 A1 20010116 (200109) A61K031-66

EP 660708 B1 20020417 (200227) EN A61K031-19

R: AT BE CH DE DK ES FR GB GR IT LI LU MC NL SE

DE 69232564 E 20020523 (200241) A61K031-19

ES 2174833 T3 20021116 (200302) A61K031-19

JP 3387498 B2 20030317 (200323) 10 A61K031-20

ADT WO 9221335 A1 WO 1992-SE333 19920519; AU 9219101 A AU

1992-19101 19920519, WO 1992-SE333 19920519; PT 100524 A

PT 1992-100524 19920527; NZ 242697 A NZ 1992-242697

19920512; JP 06508123 W JP 1992-511338 19920519, WO

1992-SE333 19920519; AU 657969 B AU 1992-19101 19920519; EP

660708 A1 EP 1992-911596 19920519, WO 1992-SE333

19920519; US 5434183 A WO 1992-SE333 19920519, US

1993-157024 19931229; SG 64864 A1 SG 1996-1678 19920519; EP

660708 B1 EP 1992-911596 19920519, WO 1992-SE333

19920519; DE 69232564 E DE 1992-632564 19920519, EP

1992-911596 19920519, WO 1992-SE333 19920519; ES 2174833 T3

EP 1992-911596 19920519; JP 3387498 B2 JP 1992-511338

19920519, WO 1992-SE333 19920519

FDT AU 9219101 A Based on WO 9221335; JP 06508123 W Based on WO 9221335; AU

657969 B Previous Publ. AU 9219101, Based on WO 9221335; EP 660708 A1

Based on WO 9221335; US 5434183 A Based on WO 9221335; EP 660708 B1 Based

on WO 9221335; DE 69232564 E Based on EP 660708, Based on WO 9221335; ES

2174833 T3 Based on EP 660708; JP 3387498 B2 Previous Publ. JP 06508123,

Based on WO 9221335

PRAI SE 1991-1642 19910530

REP DE 3347269; DE 3721137; SE 8705122; US 4820731; WO 8600523; WO 8702247

IC ICM A23D007-02; A61K031-19; A61K031-20; A61K031-66; B01F003-08;

C08L091-00

ICS A23D005-00; A23D007-00; A23D009-00; A23J007-00; **A23L001-30**;  
A61K009-10; A61K009-107; A61K009-127; A61K031-23; A61K037-22;  
A61P029-00; A61P037-06; B01F017-00; B01F017-14; B01F017-34;  
C07F009-10

AB WO 9221335 A UPAB: 19931118

In an emulsion containing vegetable **oil** and/or marine **oil**,  
an aqueous phase and **phospholipid** (I). (I) is of marine and/or  
synthetic origin and contains at least 30 weight % **omega-3**  
- **fatty acids** (03FA).

Also new are (I) use of such (I) as therapeutic agents and (2) mono-,  
bi- and/or multi-layered vesicles containing such (I). Specifically, 03FA are  
DHA (**docosahexaenoic acid**) and EPA (**eicosapentaenoic acid**). The  
emulsion contains 0.5-40 weight volume % **oil** and 0.1-30 weight % volume %  
(I), opt. also one or more biologically active cpds.

USE/ADVANTAGE - The new nutritional emulsions (for infant or total  
parenteral nutrition) result in lower blood levels **triglycerides**  
and cholesterol than similar emulsions containing the same amount of 03FA as  
**fish oil**. (I) also have **antiinflammatory**  
and/or immunosuppressant effects (especially for treating rheumatoid arthritis  
and **sepsis**) and affect normal brain and retinal development and  
function. Vesicles are useful as carriers for drugs or diagnostic reagents  
and opt. include a receptor-specific ligand to improve targetting. When  
admin. as (I), 03FA (and also **omega-6 fatty acids** present in the vegetable **oil** component) are  
incorporated at higher levels into biological membranes  
Dwg.0/7

FS CPI

FA AB; DCN

MC CPI: **B04-B01B**; B04-B01C; B05-B01P; B10-C04E; **B12-A01**;  
B12-D02; B12-D02B; B12-D07; B12-H03; B12-M03; D03-C; D03-F07;  
**D03-H01T**; E05-G09C; E05-G09D

ABEQ US 5434183 A UPAB: 19950904

An emulsion comprising vegetable and/or marine **oil**, aq. phase  
and **phospholipids** as emulsifier contains omega fatty acids, viz,  
DHA and EPA, in amt. 30+ % wt.. Amt. **oil** is 0.5-40 % wt. and  
**phospholipids** 0.1-30 % wt.. Other bioactive components may be  
present. The form may be as mono-, bi- and/or multi-layered vesicles.

USE - Compsns. give low blood **triglyceride** and cholesterol  
levels and are used in treatment of **inflammatory** and  
immunosuppressive disorder e.g. rheumatoid arthritis and **sepsis**  
and also have effect on normal brain and retina development.  
Dwg.0/7

L142 ANSWER 12 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1992-378172 [46] WPIX

DNC C1992-168000

TI Immuno-potentiating prod. containing **omega-3 fatty acid**, ester, glycerine, **phospholipid** - when administered  
in early post-operative recovery, increases NK cell activity preventing  
complications i.e. infection.

DC B05 D13

PA (NISS) NISSHIN FLOUR MILLING CO

CYC 1

PI JP 04279523 A 19921005 (199246)\* 4 A61K031-23 <--

ADT JP 04279523 A JP 1991-65717 19910111

PRAI JP 1991-65717 19910111

IC ICM A61K031-23

ICS A23D009-00; A61K037-22

ICA **A23L001-30**

AB JP 04279523 A UPAB: 19931116

Product contains **omega-3 fatty acid**  
or ester, **phospholipid** or **glyceride** that have  
**omega-3 fatty acid** as fatty

**acid** components. In composed **fatty acid**, content as **omega-3 fatty acid** is regulated to 1-8 fold against **omega-6 fatty acid**.

USE/ADVANTAGE - Prod. increases activity of NK cell. By administration in an early stage of post operation, decrease of immunopotency can be prevented and complication (infection) can be prevented. NK cell shows phagocytosis against infused foreign body (especially virus) at once, different from T cell or B cell. It keeps complication (infection) at a minimum, until T cell differentiates, and antibody is produced from B cell. The fat processing product is per intestine preparation as NK cell activity increasing preparation It can be administered as capsules, liquids, granules, fine subtilise, powders, suspensions, emulsions, etc. It can be administered concomitantly in common per intestine nutrient. Daily dose for an adult is 0.1 - 50 g pref. 0.3 - 40 g as **omega-3 fatty acid**. It can also be used by adding with common nutritive food.

Dwg.0/0

FS CPI

FA AB; DCN

MC CPI: **B04-B01B**; B10-C04E; B10-G02; **B12-A01**;  
**B12-A06**; B12-D02B; B12-J01; D03-C; **D03-H01T**

L142 ANSWER 13 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1990-194842 [26] WPIX

DNC C1990-084229

TI Dietetic compsn. or pharmaceutical containing blackcurrant oil - for preventing **inflammation**, platelet aggregation and metastatic cancer.

DC B04 D13

IN BUCHANAN, M; CROZIER-WILLI, G; FLEITH, M; CROZIERWIL, G

PA (NEST) SOC PROD NESTLE SA; (WILL-I) CROZIER-WILLI G; (NEST) NESTEC SA

CYC 20

PI EP 374591 A 19900627 (199026)\* <--

R: AT BE CH DE ES FR GB IT LI LU NL SE

PT 92690 A 19900629 (199031) <--

AU 8945547 A 19900628 (199034) <--

NO 8905183 A 19900625 (199034) <--

CA 2004332 A 19900623 (199036) <--

JP 02221228 A 19900904 (199041) <--

ZA 8909632 A 19900926 (199044) <--

CH 676909 A 19910328 (199116) <--

US 5141958 A 19920825 (199237) 7 A61K031-20 <--

EP 374591 B1 19921104 (199245) FR 17 A61K037-22 <--

R: AT BE CH DE ES FR GB IT LI LU NL SE

DE 68903378 E 19921210 (199251) A61K037-22 <--

US 5234952 A 19930810 (199333)# 7 A61K031-20 <--

ES 2045363 T3 19940116 (199407) A61K037-22 <--

IE 62603 B 19950222 (199519) A61K031-19 <--

CA 2004332 C 20000718 (200045) EN A61K035-00

JP 3284132 B2 20020520 (200236) 10 A61K035-78

ADT EP 374591 A EP 1989-122506 19891206; JP 02221228 A JP

1989-334614 19891222; ZA 8909632 A ZA 1989-9632 19891215;

US 5141958 A US 1989-446130 19891205; EP 374591 B1 EP

1989-122506 19891206; DE 68903378 E DE 1989-603378 19891206

, EP 1989-122506 19891206; US 5234952 A Div ex US

1989-446130 19891205, US 1992-890542 19920528; ES 2045363

T3 EP 1989-122506 19891206; IE 62603 B IE 1989-3878

19891205; CA 2004332 C CA 1989-2004332 19891130; JP 3284132

B2 JP 1989-334614 19891222

FDT DE 68903378 E Based on EP 374591; US 5234952 A Div ex US 5141958; ES

2045363 T3 Based on EP 374591; JP 3284132 B2 Previous Publ. JP 02221228

PRAI CH 1988-4790 19881223

REP EP 92076; EP 92085; EP 902076

IC ICM A61K031-19; A61K031-20; A61K035-00; A61K035-78; A61K037-22

ICS **A23L001-21**; A61K031-215; A61K031-23; A61P009-00

AB EP 374591 A UPAB: 19930928

Use of blackcurrant **oil** to prepare a dietetic compsn. or pharmaceutical capable of improving the bioavailability of **dihomogamma linolenic** and **eicosapentaenoic** acid in relation to the bioavailability of **arachidonic** acid, is new.

USE/ADVANTAGE - The dietetic compsn. or pharmaceutical can be used to prevent (a) **inflammatory** conditions, (b) platelet aggregation and thromboses and (c) the proliferation and spreading of metastatic cancer. Dosage is 1-25g blackcurrant **oil**/day, especially 2-5g of blackcurrant pip **oil**/day.

0/0

FS CPI

FA AB

MC CPI: B04-B01C1; B12-D07; B12-G07; B12-H02; B12-J01; **D03-H01T**

ABEQ EP 374591 B UPAB: 19930928

The use of a blackcurrent **lipid** for the preparation of a dietetic or pharmaceutical composition for promoting the bioavailability of **dihomogammalinolenic** acid and **eicosapentaenoic** acid over the bioavailability of **arachidonic** acid.

0/0

ABEQ US 5141958 A UPAB: 19930928

A new treatment for cancer metastases comprises admin. **lipids** from blackcurrant seed to selectively reduce inter cell adhesion. Dosage is 1-25 g/day of **lipids**. Pref. 2-5 g/day blackcurrant seed **oil** po., p.e. or rectally, opt. as dietetic compsn.

ADVANTAGE - EPA and DHLA are provided while bioavailability of AA is depressed, without the disadvantages of ingesting **fish oil**

0/0

ABEQ US 5234952 A UPAB: 19931119

Thrombosis prevention comprises admin. of **lipids** obtd. from blackcurrent seeds in an effective amt. to reduce thrombogenicity of blood vessels. The **lipid** is extracted blackcurrent seed **oil**, a mixt. of fatty acids obtd. by the hydrolysis or fractionation of the **oil**, a pharmaceutically acceptable salt of the fatty acids, a mixt. of the fatty acids with glycerol or a mixt. of all of them. A fat-soluble antioxidant may be included e.g. tocopherols, ascorbic acid, etc..

USE/ADVANTAGE - Used for prevention of **inflammatory** diseases, platelet adhesion, platelet aggregation, thromboses, etc., for the proliferation and dissemination of cancerous metastases and for inhibiting adhesion of immune cells. The compsns. obtd. from the **lipid** promote the bioavailability of dihomagammalinolenic acid and **eicosapentaenoic** acid over **arachidonic** acid. Admin. is oral, enteral, rectal or parenteral.

Dosage is e.g. 1-25g **lipid**, pref. 2-5g **oil** per day in a single dose or pref. 2 or 3 doses.

Dwg.0/0

L142 ANSWER 14 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1989-099868 [13] WPIX

CR 1986-042032 [06]

DNC C1989-044116

TI New tri **glyceride(s)** and dietary supplements - containing omega-3 and medium-chain fatty acid components.

DC B05 C03 D13 E13

IN BABAYAN, V K; BISTRIAN, B R; BLACKBURN, G L; MASCIOLI, E A

PA (NEWE-N) NEW ENGLAND DEACONESS HOSPITAL

CYC 15

PI WO 8902275 A 19890323 (198913)\* EN 25 <--  
 RW: AT BE CH DE FR GB IT LI LU NL SE  
 W: AU JP  
 AU 8825354 A 19890417 (198929) <--  
 US 4871768 A 19891003 (198949) 6 <--  
 EP 374188 A 19900627 (199026) <--  
 R: AT BE CH DE FR GB IT LI LU NL SE  
 JP 02502010 W 19900705 (199033) <--  
 EP 374188 B1 19931103 (199344) EN 10 A61K037-22 <--  
 R: AT BE CH DE FR GB IT LI LU NL SE  
 DE 3885462 G 19931209 (199350) A61K037-22 <--  
 CA 1324155 C 19931109 (199351) C11C003-08 <--  
 EP 374188 A4 19900926 (199513) <--  
 JP 2722229 B2 19980304 (199814) 6 C07C069-587 <--

ADT WO 8902275 A WO 1988-US3037 19880831; US 4871768 A US  
 1987-92438 19870903; EP 374188 A EP 1988-908842 19880831;  
 JP 02502010 W JP 1988-508081 19880831; EP 374188 B1 EP  
 1988-908842 19880831, WO 1988-US3037 19880831; DE 3885462 G  
 DE 1988-3885462 19880831, EP 1988-908842 19880831,  
 WO 1988-US3037 19880831; CA 1324155 C CA 1988-576566  
 19880906; EP 374188 A4 EP 1988-908842 ; JP 2722229 B2 JP  
 1988-508081 19880831, WO 1988-US3037 19880831

FDT EP 374188 B1 Based on WO 8902275; DE 3885462 G Based on EP 374188, Based  
 on WO 8902275; JP 2722229 B2 Previous Publ. JP 02502010, Based on WO  
 8902275

PRAI US 1984-630732 19840712; US 1987-92438  
 19870903

REP 2.Jnl.Ref; US 4272548; EP 120169

IC A23D009-00; A23L001-30; A61K031-22; A61K037-22; C07C069-58  
 ICM A61K037-22; C07C069-587; C11C003-08  
 ICS A23D009-00; A23D009-007; A23L001-30; A61K031-22;  
 A61K031-23; A61K035-60; A61K038-00; C07C069-58; C11C003-10

AB WO 8902275 A UPAB: 19950412  
 New synthetic **triglycerides** (I) contain at least one omega-3  
 fatty acid gp. and at least one caprylic and/or capric acid gp. Also  
 claimed are dietary supplements, (II) containing 10-40% of an 'oily  
 lipid fraction' comprising 10-90 weight% medium-chain  
**triglycerides** and 10-50 weight% of omega-3 fatty acids.  
 (I) are of formula R1OCH2-CHOR2-CH2OR3, where R1 and R3 = caprylic  
 and/or capric acid gps. and R2 = an omega-3 fatty acid gp. (I) are prepared  
 by conventional transesterification, especially using a **fish**  
**oil** as omega-3 fatty acid source.  
 (II) may also contain 1-2 weight% lecithin, 1-3 weight% of an osmolarity  
 modifier (especially glycerol) and 0-90% of an **oil** containing omega-9  
 fatty acids, especially a high-oleic-acid vegetable **oil**.  
 USE/ADVANTAGE - (I) and (II) are useful for animal and especially human  
 nutrition. They promote resistance to infection and cardiovascular  
 problems, esp., in patients suffering from bacterial infections, surgical  
 trauma, burns, malnutrition, ageing or cancer and those with secondary  
 immunosuppression due to chemotherapy or diabetes.  
 Dwg.0/0  
 Dwg.0/0

FS CPI  
 FA AB; DCN

MC CPI: B10-C02; B10-E04C; B12-A01; B12-D02A; B12-F01C; B12-J01;  
 B12-L09; C10-C02; C10-E04C; C12-A01; C12-D02A; C12-F01C;  
 C12-J01; C12-L09; D03-G01; D03-H01T

ABEQ US 4871768 A UPAB: 19930923  
 New synthetic **triglyceride** (I) has glycerol backbone with 3  
 fatty acids attached. Acids are selected from a 1st. gp. of omega3 fatty  
 acids and a 2nd gp. of caprylic acid and/or capric. At least one fatty  
 acid is from each of gp.s 1 and 2, pref. 1 from 2nd. these pref.being  
 bound to non-adjacent C atoms, the whole comprising a rearranged

structured lipid. The omega3 acids may be from plant. plankton, fungal, or fish oils esp. menhaden, salmon, anchovy or herring oil. ( New method of minimising the effects of infection and subsequent infection comprises admin. e.g. as supplement or as total p.e. nutrition, of 10-80 wt.% Oily fraction of (I) or 10-40 % wt. oily fraction having 10-90% of (I), and also 1-2% phospholipid emulsifier and 1-3% osmolarity modifier (glycerol).

ADVANTAGE - Good absorption of calories promotes resistance to , and mimimises effects of infection in vulnerable patients. (I) does not simulate insulin release or block reticuloendothelial system.

ABEQ EP 374188 B UPAB: 19931213

A synthetic triglyceride comprising a glycerol backbone having three fatty acids attached thereto, said fatty acids being selected from a first group consisting of omega 3 fatty acids, and a second group consisting of caprylic acid, and/or capric acid, whereby at least one of said fatty acids is selected from said first group and at least one (for example two) of said fatty acids is selected from said second group, and whereby if two of said fatty acids are selected from said second group, said two fatty acids are bound to adjacent carbon atoms of said glycerol backbone.

Dwg.0/0

L142 ANSWER 15 OF 15 WPIX COPYRIGHT 2004 THOMSON DERWENT on STN

AN 1986-042032 [06] WPIX

CR 1989-099868 [13]

DNC C1986-017870

TI Minimising effects of infection - by administering a diet rich in omega-3 fatty acids.

DC B05 C03 D13

IN BABAYAN, V K; BISTRIAN, B R; BLACKBURN, G L; MASCIOLI, E

PA (MASC-I) MASCIOLI E A; (NEWE-N) NEW ENGLAND DEACONS

CYC 14

PI WO 8600523 A 19860130 (198606)\* EN 23 <--

RW: AT BE CH DE FR GB IT LU NL SE

W: AU JP

AU 8546326 A 19860210 (198619) <--

EP 188573 A 19860730 (198631) EN <--

R: AT BE CH DE FR GB IT LI LU NL SE

JP 61502816 W 19861204 (198703) <--

US 4752618 A 19880621 (198827) <--

US 4820731 A 19890411 (198917) <--

AU 9063751 A 19910509 (199126) <--

EP 188573 B 19920318 (199212) 13 <--

R: AT BE CH DE FR GB IT LI LU NL SE

DE 3585681 G 19920423 (199218) <--

JP 07059508 B2 19950628 (199530) 7 A61K031-20 <--

ADT WO 8600523 A WO 1985-US1332 19850711; EP 188573 A EP 1985-903749 19850711; JP 61502816 W JP 1985-503321 19850711 ; US 4752618 A US 1984-630732 19840712; US 4820731 A US 1989-207122 19890615; EP 188573 B EP 1985-903749 19850711; JP 07059508 B2 JP 1985-503321 19850711, WO 1985-US1332 19850711

FDT JP 07059508 B2 Based on JP 61502816, Based on WO 8600523

PRAI US 1984-630732 19840712; US 1987-92438

19870903; US 1988-207122 19880615

REP 4.Jnl.Ref; DE 3213744; SSR870520; EP 106571

IC A23D007-00; A23L001-30; A61K031-23; A61K035-60

ICM A61K031-20

ICS A23D007-00; A23L001-30; A61K031-23; A61K035-60

ICA A23L001-29

AB WO 8600523 A UPAB: 19950412

A method of minimising the effects of infection comprises administering a diet rich in omega-3 fatty acids.



These are pref. **oils** derived from herring, anchovy, cod and menhaden. A pref. dietary supplement comprises 10-20 weight% oily fraction containing **omega-3 fatty acids**, 1-2 weight% of an emulsifier (e.g. egg yolks, **phospholipids** and soybean **phospholipids**), 1-3 weight% of an osmolality modifier (e.g. glycerin) and sterile water.

USE/ADVANTAGE - By replacing the predominantly **omega-6 fatty acid** containing **oils** with **omega-3 fatty acid**-containing **oils**, the levels of type 2 prostaglandins is reduced and the levels of type 3 prostaglandins is increased. Patients infected with wound infections, empyemas, bacteremias, abscesses and **septicemias**, may be treated. Patients at high risk of infection are also treated e.g. patient with secondary immunosuppression due to chemotherapy or diabetes mellitus, protein-malnourished patients and patients undergoing abdominal surgery.

Dwg.0/0

Dwg.0/0

FS CPI

FA AB

MC CPI: **B04-B01B**; **B04-B01C2**; **B12-A01**; **B12-A02C**;  
**B12-A06**; **B12-A07**; **B12-B04**; **B12-J01**; **C04-B01B**;  
**C04-B01C2**; **C12-A01**; **C12-A02C**; **C12-A06**; **C12-A07**;  
**C12-B04**; **C12-J01**; **D03-H01T**

ABEQ DE 3585681 G UPAB: 19930922

A method of minimising the effects of infection comprises administering a diet rich in **omega-3 fatty acids**.

These are pref. **oils** derived from herring, anchovy, cod and menhaden. A pref. dietary supplement comprises 10-20 wt.% oily fraction contg. **omega-3 fatty acids**, 1-2 wt.% of an emulsifier (e.g. egg yolks, **phospholipids** and soybean **phospholipids**), 1-3 wt.% of an osmolality modifier (e.g. glycerin) and sterile water.

USE/ADVANTAGE - By replacing the predominantly **omega-6 fatty acid** contg. **oils** with **omega-3 fatty acid**-contg. **oils**, the levels of type 2 prostaglandins is reduced and the levels of type 3 prostaglandins is increased. Patients infected with wound infections, empyemas, bacteremias, abscesses and **septicemias**, may be treated. Patients at high risk of infection are also treated e.g. patient with secondary immunosuppression due to chemotherapy or diabetes mellitus, protein-malnourished patients and patients undergoing abdominal surgery.

ABEQ EP 188573 B UPAB: 19930922

The use of **omega3 fatty acids** for the manufacture of a dietary material for minimising the effects of infection in animals (e.g.

humans) other than avians.

ABEQ US 4752618 A UPAB: 19930922

Infection and subsequent infection in high risk patients is minimised by administering a diet which is controlled in its caloric and **fatty acid** intake. Diet contains 10-20 wt.% of mixt. of **fatty acid**-contg. **oils** which comprise **omega 6 fatty acids**, and 10-90% of **omega 3 fatty acids** each or their prim **fatty acids**. Pref. **omega 3 oils** comprise **oils** derived from herring, anchovy or menhaden.

USE - In treatment of patients infected with wound infections, empyemae, bacteremias, abscesses, or **septicemias**, as a result of sec. immunosuppression due to chemotherapy or diabetes mellitus, protein-malnourishment or abdominal surgery.

ABEQ US 4820731 A UPAB: 19930922

Dietary supplement comprises higher unsatd. **fatty acid**

**oils** (at least 10 emulsifier (1-2 wt. %); glycerol (1-3 wt. %) as an osmolality modifier; and water. The oily component comprises a mixt. of **omega-3-fatty acid oils** (10-90 wt.%) and **omega-6-fatty acid oils** as the major components.

Pref. emulsifier are egg yolk **phospholipids** and soya bean **phospholipids**.

USE - The prods. are suitable for parenteral administration.

=> d his

(FILE 'HOME' ENTERED AT 09:35:24 ON 31 AUG 2004)  
SET COST OFF

FILE 'HCAPLUS' ENTERED AT 09:35:33 ON 31 AUG 2004

L1 1 S (WO2000-EP8731 OR EP99-118173)/AP,PRN  
E NESTLE/PA,CS  
L2 2617 S NESTLE?/PA,CS  
E TURINI M/AU  
L3 25 S E3-E6  
E ROESSLE C/AU  
L4 8 S E3,E4  
E ROSSLE C/AU  
L5 7 S E3,E4  
E BREUILLE D/AU  
L6 34 S E3,E4  
E CROZIER W/AU  
L7 5 S E3,E9,E10  
E WILLI G/AU  
E CROZIER G/AU  
L8 24 S E3-E8  
E FINOT P/AU  
L9 80 S E4-E7  
E RICHELLE M/AU  
L10 45 S E3,E4,E8  
E DUTOT G/AU  
L11 11 S E3,E5

FILE 'REGISTRY' ENTERED AT 09:45:51 ON 31 AUG 2004

L12 6 S (LINOLEIC ACID OR  $\Gamma$ -LINOLENIC ACID OR DIHOMO- $\Gamma$ -LI  
L13 9 S (EICOSAPENTAENOIC ACID OR DOCOSAPENTAENOIC ACID OR DOCOSAHEXA  
L14 1 S 92661-11-5  
L15 7 S L12,L14  
L16 1 S DIHOMO- $\Gamma$ -LINOLEINIC ACID/CN  
L17 7 S L15,L16

FILE 'HCAPLUS' ENTERED AT 09:52:10 ON 31 AUG 2004

L18 52030 S L17  
L19 1471 S L17(L) FFD/RL  
L20 1280 S L17(L) THU/RL  
L21 20593 S L18 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
L22 63109 S (ARACHIDONIC OR LINOLEIC OR GAMMA LINOLENIC OR DIHOMO GAMMA()  
L23 24880 S L22 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
E FATTY ACIDS/CT  
E FATTY ACIDS (L) N/CT  
E FATTY ACIDS (L) OMEGA/CT  
E FATTY ACIDS (L) POLYUNSAT/CT  
L24 2403 S E13,E16  
L25 2519 S FATTY ACID?/CT (L) (N6 OR (N OR OMEGA) () 6 OR OMEGA6)  
L26 276 S L24,L25 (L) FFD/RL  
L27 168 S L24,L25 (L) THU/RL  
L28 2167 S L24,L25 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?

L29 30232 S L19,L20,L21,L23,L26,L27,L28  
 L30 26049 S L13  
 L31 1830 S L13 (L) FFD/RL  
 L32 1316 S L13 (L) THU/RL  
 L33 14009 S L30 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
 L34 12812 S (EICOSAPENTAENOIC OR DOCOSAPENTAENOIC OR DOCOSAHEXAENOIC OR A  
 L35 8135 S L34 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
 E FATTY ACIDS (L) POLYUNSAT/CT  
 L36 5489 S E12,E14,E15  
 L37 5703 S FATTY ACIDS?/CT (L) (N3 OR (N OR OMEGA) ()3 OR OMEGA3)  
 L38 855 S L36,L37 (L) FFD/RL  
 L39 654 S L36,L37 (L) THU/RL  
 L40 5131 S L36,L37 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT?  
 L41 17717 S L31,L32,L33,L35,L38,L39,L40  
 L42 11526 S L29 AND L41  
 L43 21471 S L18-L29 AND L30-L41  
 L44 5431 S L43 AND LIPID#/CW  
 E MEDIUM CHAIN TRIGLYCERIDE/CT  
 E E11+ALL  
 L45 1538 S E2  
 L46 2489 S ((MED OR MEDIUM OR M) ()CHAIN) (L) (GLYCERIDE OR TRIGLYCERIDE)  
 E FISH OIL/CT  
 E E5+ALL  
 L47 4257 S E2  
 L48 1570 S L47 (L) (FFD/RL OR THU/RL)  
 L49 3744 S L47 AND (FOOD? OR FEED? OR DIET? OR NUTRI? OR PHARMACEUT? OR  
 L50 715 S L47-L49 AND LIPID#/CW  
 L51 85 S L47-L49 AND L45,L46  
 L52 5950 S L44,L50,L51  
 E SEPSIS/CT  
 L53 7317 S E3,E5-E10  
 E E8+ALL  
 L54 8136 S E1,E2,E3  
 E E3+ALL  
 L55 3750 S E4+NT  
 E E3+ALL  
 L56 10652 S E3+NT  
 E SHOCK/CT  
 L57 4729 S E4-E12  
 E E4+ALL  
 L58 13891 S E8,E9,E7+NT  
 E E26+ALL  
 L59 6857 S E3,E2+NT  
 L60 8 S L52 AND L53-L59  
 L61 152 S L1-L11 AND L18-L60  
 L62 17 S L61 AND L47-L49  
 L63 55 S L61 AND L42,L43  
 L64 30 S L63 AND LIPID  
 L65 41 S L62,L64  
 L66 23 S L62,L63 NOT L65  
 L67 8 S L52 AND ?SEPSI?  
 L68 13 S L52 AND ?SEPTI?  
 L69 17 S L60,L67,L68  
 L70 1 S L61 AND L69  
 L71 14 S L69 AND (PY<=1999 OR PRY<=1999 OR AY<=1999)  
 L72 3 S L69 NOT L70,L71  
 L73 1 S L72 AND SEPTIC RAT  
 SEL DN AN L71 2 6 12 13  
 L74 10 S L71 NOT E1-E12  
 L75 11 S L73,L74,L70  
 L76 11 S L75 AND (N6 OR N 6 OR OMEGA6 OR OMEGA 6 OR N3 OR N 3 OR OMEGA  
 L77 8 S L76 AND (LINOLEIC OR ?LINOLENIC? OR ARACHINDONIC OR DOCOSADIE  
 L78 11 S L75-L77 AND L1-L11,L18-L77

FILE 'HCAPLUS' ENTERED AT 10:26:26 ON 31 AUG 2004

FILE 'MEDLINE' ENTERED AT 10:37:40 ON 31 AUG 2004

L79 14314 S L17  
L80 40760 S L22  
L81 4507 S (N6 OR OMEGA6 OR (N OR OMEGA) () 6) (L) FATTY ACID  
L82 43258 S L79-L81  
L83 3226 S L13  
L84 6867 S L34  
L85 7356 S (N3 OR OMEGA3 OR (N OR OMEGA) () 3) (L) FATTY ACID  
L86 11440 S L83-L85  
L87 4872 S FISH OIL  
E FISH OIL/CT  
E E4+ALL  
L88 9187 S E4+NT  
E FATTY ACIDS/CT  
L89 6700 S E88+NT  
L90 8276 S E112+NT  
L91 43258 S L82,L90  
L92 10055 S L85,L89  
L93 5250 S L91 AND L92  
L94 10919 S L88,L93  
E MEDIUM CHAIN/CT  
E TRIGLYCERIDE/CT  
E E5+ALL  
E E3+ALL  
L95 1344 S E3+NT AND L94  
L96 4273 S LIPID AND L94  
L97 4758 S L95,L96  
E SEPSIS/CT  
E E3+ALL  
L98 52791 S E4+NT  
E SHOCK/CT  
E E3+ALL  
L99 35591 S E5+NT  
L100 17 S L97 AND L98,L99  
L101 15 S L97 AND SHOCK  
L102 28 S L97 AND (?SEPSIS? OR ?SEPTIC?)  
L103 37 S L100-L102  
L104 25 S L103 AND PY<=1999  
SEL DN AN 1 5 9 10 11  
L105 20 S L104 NOT E1-E10  
L106 12 S L103 NOT L104  
SEL DN AN 8 10  
L107 10 S L106 NOT E11-E14  
L108 30 S L105,L107 AND L79-L107  
L109 30 S L108 AND (OMEGA? OR N6 OR N3 OR FATTY ACID OR ?GLYCERIDE? OR

FILE 'MEDLINE' ENTERED AT 10:52:08 ON 31 AUG 2004

FILE 'WPIX' ENTERED AT 10:52:28 ON 31 AUG 2004

L110 1 S L1  
L111 27932 S LIPID/BIX OR (B04-B01B OR C04-B01B)/MC  
L112 308 S L111 AND FISH OIL/BIX  
L113 26 S L111 AND ((N3 OR OMEGA3) (L) FATTY ACID)/BIX  
L114 305 S L111 AND (((N OR OMEGA) () 3) (L) FATTY ACID)/BIX  
L115 510 S L111 AND (EICOSAPENTAEN? OR DOCOSAPENTAEN? OR DOCOSAHEXAEN? O  
L116 10 S L111 AND ((N6 OR OMEGA6) (L) FATTY ACID)/BIX  
L117 140 S L111 AND (((N OR OMEGA) () 6) (L) FATTY ACID)/BIX  
L118 890 S L111 AND (LINOLEIC OR GAMMA LINOLENIC OR DIHOMO GAMMALINOLENIC  
L119 344 S L113,L114,L115 AND L116,L117,L118  
L120 601 S L112,L119

L121 11 S L120 AND (SEPSIS OR SEPTICEM? OR SEPTICAEM?)/BIX  
L122 18 S L120 AND (B14-S06 OR C14-S06 OR B12-A01 OR C12-A01 OR B12-A06  
L123 104 S L120 AND (P220 OR P420)/M0,M1,M2,M3,M4,M5,M6  
L124 118 S L120 AND ?INFLAM?/BIX  
L125 6 S L120 AND SHOCK/BIX  
L126 39 S L121-L125 AND D03-H01T?/MC  
L127 36 S L121-L125 AND A23L001/IPC  
L128 53 S L126,L127  
L129 19 S L128 AND PY<=1999  
L130 28 S L128 AND PRY<=1999  
L131 27 S L128 AND AY<=1999  
L132 28 S L129-L131  
L133 25 S L128 NOT L132  
L134 22 S L132 AND (SHOCK OR ?INFLAM?)/BIX  
L135 8 S L132 AND L121,L122  
L136 26 S L134,L135  
SEL DN AN 2 3 4 5 7 8 11 15 16 22 25  
L137 15 S L136 NOT E15-E37  
L138 15 S L110,L137  
L139 13 S L138 AND ?LIPID?/BIX  
L140 8 S L138 AND ?GLYCERID?/BIX  
L141 9 S L138 AND OIL/BIX  
L142 15 S L138-L141

FILE 'WPIX' ENTERED AT 11:22:07 ON 31 AUG 2004

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